

=> d que stat

L8 12 SEA FILE=REGISTRY ABB=ON (ACRIVASTINE OR ASTEMIZOLE OR AZELASTINE OR CETIRIZINE OR EBASTINE OR EPINASTINE OR FEXOFENADINE OR DESLORATADINE OR LORATADINE OR MIZOLASTINE OR NORASTEMIZOLE OR PROMETAZINE OR TERFENADINE)/CN

L9 1 SEA FILE=REGISTRY ABB=ON PROMETHAZINE/CN

L10 13 SEA FILE=REGISTRY ABB=ON L8 OR L9

L14 836 SEA FILE=HCAPLUS ABB=ON (?HISTAMINE?(W)?RECEPT?(W)?ANTAGON?)

L15 91 SEA FILE=HCAPLUS ABB=ON L14 AND (L10 OR ?ACRIVASTINE? OR ?ASTEMIZOLE? OR ?AZELASTINE? OR ?CETIRIZINE? OR ?EBASTINE? OR ?EPINASTINE? OR ?FEXOFENADINE? OR ?DESLORATADINE? OR ?LORATADINE? OR ?MIZOLASTINE? OR ?NORASTEMIZOLE? OR ?PROMETAZINE? OR ?TERFENADINE?)

L16 91 SEA FILE=HCAPLUS ABB=ON L14 AND L15

L18 13 SEA FILE=HCAPLUS ABB=ON L16 AND ?RELEAS?

L19 160 SEA FILE=HCAPLUS ABB=ON L16 OR ?OSMATIC?

L20 18 SEA FILE=HCAPLUS ABB=ON L19 AND ?RELEAS?

L21 18 SEA FILE=HCAPLUS ABB=ON L18 OR L20

L26 14 SEA FILE=HCAPLUS ABB=ON L21 AND (PRD<20030715 OR PD<20030715)

L29 115 SEA FILE=USPATFULL ABB=ON L21 AND (PRD<20030715 OR PD<20030715)

L30 55 SEA FILE=USPATFULL ABB=ON L29 AND (?OSMATIC? OR ?OSMOTIC?)

L31 2 SEA FILE=USPATFULL ABB=ON L30 AND ?DUAL?(3A)?RELEAS?

L32 16 DUP REMOV L31 L26 (0 DUPLICATES REMOVED)

=> d ibib abs hitstr l32 1-16

L32 ANSWER 1 OF 16 USPATFULL on STN

ACCESSION NUMBER: 2003:112567 USPATFULL

TITLE: Pharmaceutical formulations and systems for improved absorption and multistage **release** of active agents

INVENTOR(S): Chen, Feng-Jing, Salt Lake City, UT, UNITED STATES
 Venkateshwaran, Srinivasan, Salt Lake City, UT, UNITED STATES
 Krill, Steven L., Park City, UT, UNITED STATES
 Patel, Mahesh V., Salt Lake City, UT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003077297	A1	20030424 <--
APPLICATION INFO.:	US 2002-74687	A1	20020211 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2001-898553, filed on 2 Jul 2001, PENDING Continuation of Ser. No. US 1999-258654, filed on 26 Feb 1999, GRANTED, Pat. No. US 6294192 Continuation-in-part of Ser. No. US 2001-877541, filed on 8 Jun 2001, PENDING Continuation-in-part of Ser. No. US 1999-345615, filed on 30 Jun 1999, GRANTED, Pat. No. US 6267985 Continuation-in-part of Ser. No. US 2001-800593, filed on 6 Mar 2001, PENDING Division of Ser. No. US 1999-447690, filed on 23 Nov 1999, GRANTED, Pat. No. US 6248363		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	REED & ASSOCIATES, 800 MENLO AVENUE, SUITE 210, MENLO PARK, CA, 94025		
NUMBER OF CLAIMS:	145		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	7 Drawing Page(s)		

LINE COUNT: 4845

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention pertains to pharmaceutical formulations and systems for delivery of active agents, wherein a first fraction of an active agent is suspended in a vehicle and a second fraction of active agent is solubilized in the vehicle, with the suspended fraction representing about 5 weight % to about 80 weight % of the active agent and

the

second fraction representing about 20 weight % to about 95 weight % of the active agent. One or more additional active agents, which may be fully solubilized, partially solubilized, or suspended, may also be present. The first and second fractions of the active agent may or may not have different **release** profiles. Generally, a significant fraction of the solubilized drug will **release** rapidly, providing for rapid onset, while the suspended drug may be formulated for delayed and/or sustained **release**.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L32 ANSWER 2 OF 16 USPATFULL on STN

ACCESSION NUMBER: 2003:64337 USPATFULL

TITLE: Drug delivery device containing oseltamivir and an H1 antagonist

INVENTOR(S): Faour, Joaquina, Buenos Aires, ARGENTINA
Vergez, Juan A., Buenos Aires, ARGENTINA

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003044457	A1	20030306	<--
	US 6605302	B2	20030812	
APPLICATION INFO.:	US 2001-907486	A1	20010717	(9)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	INNOVAR, LLC, P O BOX 250647, PLANO, TX, 75025			
NUMBER OF CLAIMS:	39			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	2 Drawing Page(s)			
LINE COUNT:	1318			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a **dual release** solid dosage form containing a first composition that **releases** oseltamivir in a controlled manner and a second composition that **releases** an H1 antagonist in a rapid and/or immediate manner. A wide range of H1 antagonist antihistamines, especially **fexofenadine** and **loratadine**, can be used in this device. Particular embodiments of the invention provide **osmotic** devices having predetermined **release** profiles. The device is useful for the treatment of respiratory congestion and other viral infection associated symptoms.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

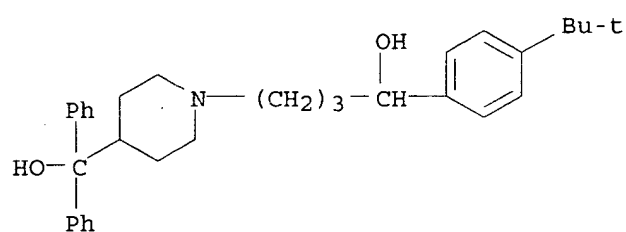
IT 60-87-7, Promethazine 50679-08-8, Terfenadine
58581-89-8, Azelastine 68844-77-9, Astemizole
75970-99-9, Norastemizole 79794-75-5, Loratadine
80012-43-7, Epinastine 83799-24-0, Fexofenadine
83881-51-0, Cetirizine 87848-99-5, Acrivastine
90729-43-4, Ebastine 108612-45-9, Mizolastine
(drug delivery device containing oseltamivir and an H1 antagonist)

RN 60-87-7 USPATFULL

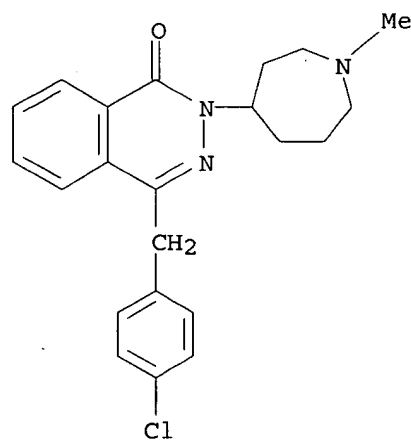
CN 10H-Phenothiazine-10-ethanamine, N,N, α -trimethyl- (9CI) (CA INDEX

CN(C)C(C)CN1Cc2ccccc2Sc3ccccc13

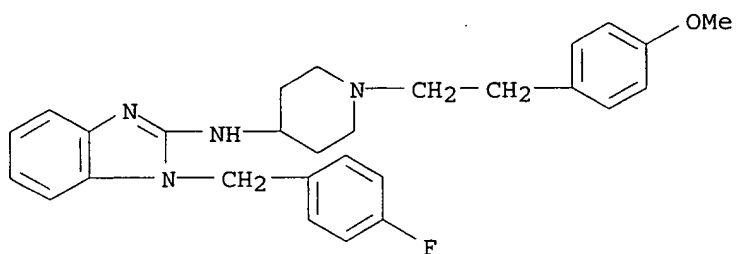
CN 1-Piperidinebutanol, α -[4-(1,1-dimethylethyl)phenyl]-4-(hydroxydiphenylmethyl)- (9CI) (CA INDEX NAME)



CN 1(2H)-Phthalazinone, 4-[(4-chlorophenyl)methyl]-2-(hexahydro-1-methyl-1H-azepin-4-yl)- (9CI) (CA INDEX NAME)

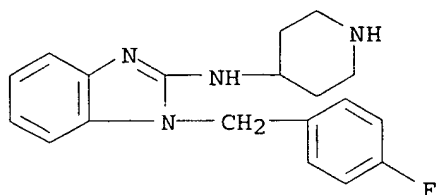


CN 1H-Benzimidazol-2-amine, 1-[(4-fluorophenyl)methyl]-N-[1-[2-(4-methoxyphenyl)ethyl]-4-piperidinyl]- (9CI) (CA INDEX NAME)



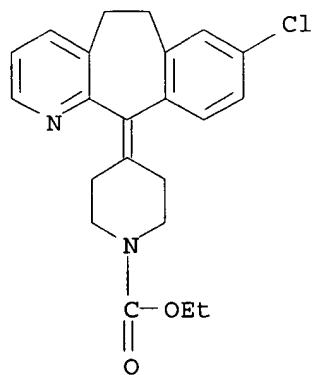
RN 75970-99-9 USPATFULL

CN 1H-Benzimidazol-2-amine, 1-[(4-fluorophenyl)methyl]-N-4-piperidinyl- (9CI)
(CA INDEX NAME)



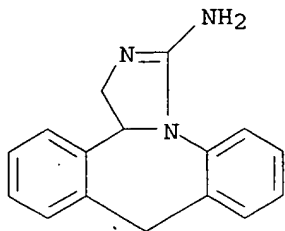
RN 79794-75-5 USPATFULL

CN 1-Piperidinecarboxylic acid, 4-(8-chloro-5,6-dihydro-11H-benzo[5,6]cyclohepta[1,2-b]pyridin-11-ylidene)-, ethyl ester (9CI) (CA INDEX NAME)



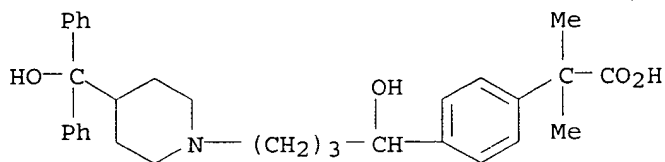
RN 80012-43-7 USPATFULL

CN 1H-Dibenz[c,f]imidazo[1,5-a]azepin-3-amine, 9,13b-dihydro- (9CI) (CA INDEX NAME)



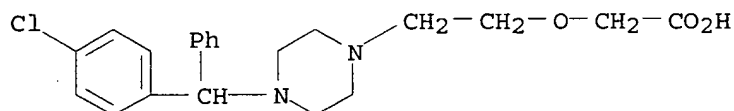
RN 83799-24-0 USPATFULL

CN Benzeneacetic acid, 4-[1-hydroxy-4-[4-(hydroxydiphenylmethyl)-1-piperidinyl]butyl]- α,α -dimethyl- (9CI) (CA INDEX NAME).



RN 83881-51-0 USPATFULL

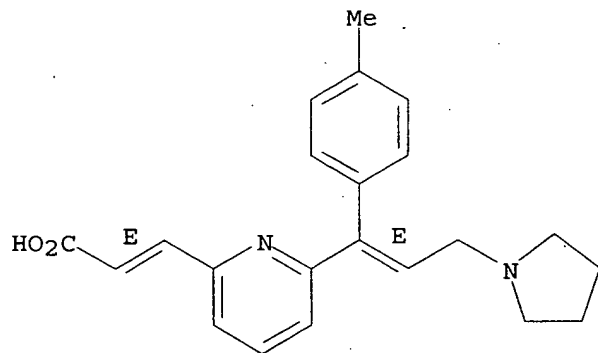
CN Acetic acid, [2-[4-[(4-chlorophenyl)phenylmethyl]-1-piperazinyl]ethoxy]- (9CI) (CA INDEX NAME)



RN 87848-99-5 USPATFULL

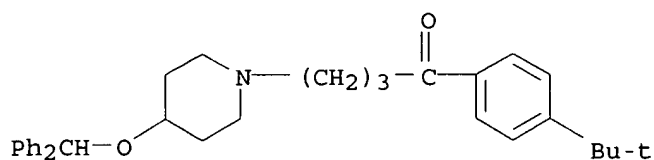
CN 2-Propenoic acid, 3-[6-[(1E)-1-(4-methylphenyl)-3-(1-pyrrolidinyl)-1-propenyl]-2-pyridinyl]-, (2E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



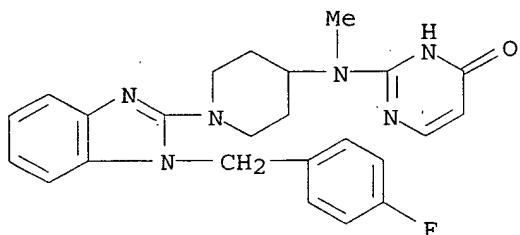
RN 90729-43-4 USPATFULL

CN 1-Butanone, 1-[4-(1,1-dimethylethyl)phenyl]-4-[4-(diphenylmethoxy)-1-piperidinyl]- (9CI) (CA INDEX NAME)



RN 108612-45-9 USPATFULL

CN 4 (1H)-Pyrimidinone, 2-[[1-[1-[(4-fluorophenyl)methyl]-1H-benzimidazol-2-yl]-4-piperidinyl]methylamino]- (9CI) (CA INDEX NAME)



L32 ANSWER 3 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:401390 HCAPLUS

DOCUMENT NUMBER: 135:151537

TITLE: Roles of mast cells and histamine in mosquito bite-induced allergic itch-associated responses in mice

AUTHOR(S): Ohtsuka, Eiji; Kawai, Sanae; Ichikawa, Tomohiro; Nojima, Hiroshi; Kitagawa, Kanji; Shirai, Yoshikazu; Kamimura, Kiyoshi; Kuraishi, Yasushi

CORPORATE SOURCE: Department of Applied Pharmacology, Faculty of Pharmaceutical Sciences, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan

SOURCE: Japanese Journal of Pharmacology (2001), 86(1), 97-105

CODEN: JJPAAZ; ISSN: 0021-5198

PUBLISHER: Japanese Pharmacological Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We investigated itch-associated responses (scratching) to mosquito bites and the role of histamine and mast cells in mosquito-induced itching in mice. Although the first bites of mosquito *Aedes albopictus* did not increase scratching, repeated bites increased scratching. The response was not diminished even after an interval of 2 mo. Similarly, repeated intradermal (i.d.) injections of salivary gland extract (SGE) from *Aedes albopictus* increased scratching after SGE injection itself and mosquito bites. The scratching peaked within 10 min and almost subsided by 60 min. The opioid antagonist naloxone (1 mg/kg, s.c.) inhibited scratching following SGE injection. Although the non-sedative H1-histamine **-receptor antagonist terfenadine** (30 mg/kg, p.o.) significantly suppressed scratching induced by histamine (100 nmol/site, i.d.) in either naive or mosquito-sensitized mice, it did not affect mosquito-induced scratching in mosquito-sensitized mice. Repeated injections of SGE increased scratching in mast cell-deficient (WBB6F1-W/Wv) mice as well as in normal (WBB6F1-+/+) littermates.

Repeated exposure to mosquito bites roughly doubled serum concns. of total IgE and IgG1, but not IgG2a. Repeated injections of SGE markedly increased plasma extravasation induced by mosquito bites and such an increase was almost completely suppressed by **terfenadine** (30 mg/kg, p.o.). The results show the presence of histamine-mediated and histamine-independent mechanisms in cutaneous itching and suggest that histamine probably **released** from mast cells does not play an important role in itching in immediate allergic reaction. Our murine model of mosquito itching may be useful for studying the mechanisms of immediate allergic itching.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 4 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1996:538056 HCAPLUS

DOCUMENT NUMBER: 125:266463

TITLE: The contribution of histamine to the action of bradykinin in the human nasal airway

AUTHOR(S): Austin, C. E.; Dear, J. W.; Neighbour, H.; Lund, V.; Foreman, J. C.

CORPORATE SOURCE: Department of Pharmacology, University College London, Gower Street, London, WC1E 6BT, UK

SOURCE: Immunopharmacology (1996), 34(2-3), 181-189

CODEN: IMMUDP; ISSN: 0162-3109

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Bradykinin, 10 to 1000 µg given by aerosol into the nasal cavity of normal, healthy volunteers, produced a dose-related increase of nasal airway resistance. Bradykinin also reduced the minimal nasal cross-sectional area (Amin), increased albumin **release** into nasal lavage fluid and increased the symptoms of nasal inflammation. Pretreatment with **cetirizine** (10 mg orally) reduced the fall in Amin induced by bradykinin, 300 µg, but not by bradykinin, 100 µg. Pretreatment of the subjects with the H1 **histamine receptor antagonist cetirizine** (10 mg, orally) or **terfenadine** (60 mg, orally) 3 h before bradykinin administration caused significant reduction of the bradykinin-induced increase in nasal airway resistance in the upper range of bradykinin doses (300-1000 µg) but not in the lower range (10-100 µg). **Cetirizine** reduced the albumin **release** into the nasal airway and the symptoms induced by bradykinin, 1000 µg. Following nasal challenge with bradykinin 300 µg or 1000 µg, no increase could be detected in the histamine content of nasal lavage fluid. Isolated human nasal cells **released** histamine in response to bradykinin, 33 and 100 µM, anti-IgE and calcium ionophore, A 23187. We conclude that the actions of bradykinin in the human nasal airway are, in part, accounted for by the **release** of histamine.

L32 ANSWER 5 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1995:622901 HCAPLUS

DOCUMENT NUMBER: 123:27494

TITLE: Protective effect of various antagonists of inflammatory mediators against paraoxon-induced pulmonary edema in the rabbit

AUTHOR(S): Delaunois, A.; Gustin, Pascal; Vargas, Mario; Ansay, Michel

CORPORATE SOURCE: Dep. of Pharmacology and Toxicology, Univ. of Liege, Liege, B4000, Belg.

SOURCE: Toxicology and Applied Pharmacology (1995),

132(2), 343-5

CODEN: TXAPA9; ISSN: 0041-008X

PUBLISHER:

Academic

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB The protective effect of some antagonists of various inflammatory mediators against paraoxon-induced increases in endothelial permeability has been investigated in isolated perfused rabbit lungs. The edema induced by paraoxon has been previously related to a chain reaction mediated by acetylcholine. Lungs were ventilated and blood-free perfused with a constant flow. Arterial and venous pressures and lung weight were continuously recorded. Endothelial permeability was evaluated by measuring the capillary filtration coefficient (Kf.c.). Paraoxon (4 + 10-4M) was injected in the perfusion circuit, in lungs with or without pretreatment with atropine, ketanserin, clonidine, morphine, indomethacin, and **terfenadine** plus cimetidine. Paraoxon induced a time-dependent increase in the Kf.c., a maximal effect being recorded 60 min after the injection. All the antagonists used as pretreatment significantly reduced the maximal effect recorded after paraoxon. These results show that muscarinic receptor antagonists, inhibitors of neuropeptides **release**, cyclooxygenase inhibitors, and 5-hydroxytryptamine and **histamine receptor antagonists** can protect the lung against the edema induced by paraoxon. This protective effect is due to inhibition of the chain reaction triggered by acetylcholine.

L32 ANSWER 6 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1996:370261 HCAPLUS

DOCUMENT NUMBER: 125:49674

TITLE: Osmotic thresholds of vasopressin **release** and thirsty sensation in healthy volunteers

AUTHOR(S): Li, Guo; Guo, Ming; Kuang, Ankun; Ding, Ting; Xu, Manyin; Chen, Jialun

CORPORATE SOURCE: Ruijin Hospital, Shanghai Second Medical Univ., Shanghai, 200025, Peop. Rep. China

SOURCE: Shanghai Dier Yike Daxue Xuebao (1994), 14(3), 233-237

CODEN: SDDXE3; ISSN: 0258-5898

PUBLISHER: Shanghai Dier Yike Daxue Xuebao Bianjibu

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB The changes of the plasma Na, plasma osmolality, plasma AVP and thirst scales in 12 healthy volunteers tested in a 2-h period of 5% NaCl infusion (0.06 mL/kg/min) were reported. Results showed that the osmoregulatory regression equation of AVP secretion and thirst sensation were plasma AVP = 0.24 [plasma osmolality (pOsm)-274] and thirst (Th) = 0.23 (pOsm-279), resp. Because both the sensitivity and thirst sensation are lower than those reported by others, it was suggested that there may be some ethnic difference in the osmoregulation of AVP secretion and thirst sensation as well as some linkage between the **osmotic** thresholds of vasopressin **release** and thirst sensation and also the influence of heredity and environmental factors.

L32 ANSWER 7 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1994:95505 HCAPLUS

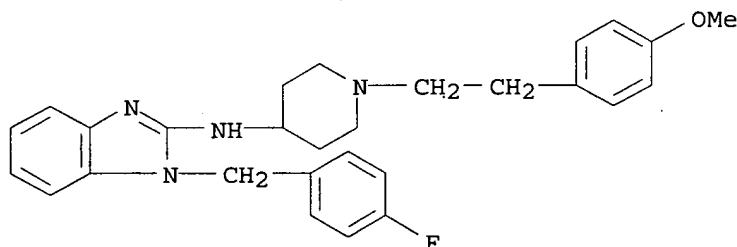
DOCUMENT NUMBER: 120:95505

TITLE: The differential effects of **histamine receptor antagonists** on morphine-

and U-50,488H-induced antinociception in the mouse

AUTHOR(S): Suzuki, Tsutomu; Takamori, Kazuaki; Takahashi, Yuki;

CORPORATE SOURCE: Narita, Minoru; Misawa, Miwa; Onodera, Kenji
Sch. Pharm., Hoshi Univ., Tokyo, 142, Japan
SOURCE: Life Sciences (1994), 54(3), 203-11
CODEN: LIFSAK; ISSN: 0024-3205
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of thioperamide, an H3 antagonist, and histamine H1 and H2 antagonists (s.c.) on morphine (s.c. or i.c.v.)- and U-50,488H (i.c.v.)-induced antinociception in male ddY mice were examined using the hot-plate (55°) test. Thioperamide significantly inhibited morphine-induced antinociception, but not U-50,488H-induced antinociception. The suppressive effect of thioperamide on morphine-induced antinociception was reversed by the H1 antagonist pyrilamine, but not by the H2 antagonist zolantidine. Pylamine significantly potentiated the antinociception induced by morphine, but not that induced by U-50,488H. Zolantidine significantly inhibited morphine-induced antinociception in a dose-dependent manner, but not U-50,488H-induced antinociception. Both **astemizole**, an H1 antagonist, and ranitidine, an H2 antagonist, which are known to barely cross the blood brain barrier, did not affect morphine-induced antinociception. These results suggest that morphine-induced antinociception may be potentiated by activation of H2 receptors and suppressed by activation of H1 receptors in the brain. Furthermore, neuronal histamine **release** induced by thioperamide may suppress morphine-induced antinociception through H1 receptors.
IT 68844-77-9, **Astemizole**
RL: BIOL (Biological study)
(analgesia from morphine and U-50,488H response to, histaminic receptors in)
RN 68844-77-9 HCAPLUS
CN 1H-Benzimidazol-2-amine, 1-[(4-fluorophenyl)methyl]-N-[1-[2-(4-methoxyphenyl)ethyl]-4-piperidinyl]- (9CI) (CA INDEX NAME)



L32 ANSWER 8 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:260831 HCAPLUS
DOCUMENT NUMBER: 120:260831
TITLE: Pharmacological study of **ebastine**, a novel histamine H1-receptor antagonist
AUTHOR(S): Yakuo, Ikuhisa; Ishii, Katsumi; Seto, Yasuhiro; Imano, Kiyomi; Takeyama, Kunihiro; Nakamura, Hideo; Karasawa, Tadahiko
CORPORATE SOURCE: Explor. Res. Lab., Dainippon Pharm. Co., Ltd., Suita, 564, Japan
SOURCE: Nippon Yakurigaku Zasshi (1994), 103(3), 121-35
CODEN: NYKZAU; ISSN: 0015-5691
DOCUMENT TYPE: Journal

LANGUAGE: Japanese

AB The anti-allergic activity of **ebastine**, a novel antihistamine, was assessed in comparison with several antihistamines. Orally administered **ebastine** dose-dependently inhibited 7-day homologous passive cutaneous anaphylaxis (PCA), exptl. allergic rhinitis and exptl. asthma in guinea pigs or rats (ED50-values were 2.17, 0.29 and 0.35 mg/kg, resp.); and its anti-allergic activity was more potent than those of **terfenadine** and **mequitazine**. Moreover, its PCA-inhibitory activity was still observed 24 h after the administration. Orally administered **ebastine** also inhibited histamine-induced skin reaction in rats (ED50: 1.10 mg/kg). In isolated guinea pig trachea, **ebastine** had no effect on histamine-induced contraction, but **carebastine**, a main metabolite of **ebastine**, inhibited this contraction (IC50: 0.12 μ M). **Carebastine** (30-100 μ M) suppressed the histamine release from rat peritoneal mast cells and human basophils. **Ebastine** at a high oral dose showed slight inhibition of the specific binding of 3H-mepyramine to the histamine H1-receptor in rat brain. This binding-inhibitory activity of **ebastine** was little more potent than that of **terfenadine**, but much less potent than those of **mequitazine** and **ketotifen**. These results indicated that **ebastine** has potent and long acting anti-allergic activity with few side effects based on the antihistaminic activity in the central nervous system. Furthermore, it was suggested that these effects of **ebastine** are due to the action of a main metabolite, **carebastine**.

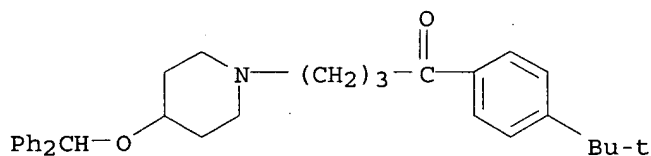
IT 90729-43-4, **Ebastine**

RL: BIOL (Biological study)

(antiallergic and antihistaminic activity of, metabolite **carebastine** in)

RN 90729-43-4 HCAPLUS

CN 1-Butanone, 1-[4-(1,1-dimethylethyl)phenyl]-4-[4-(diphenylmethoxy)-1-piperidinyl]- (9CI) (CA INDEX NAME)



L32 ANSWER 9 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1993:463711 HCAPLUS

DOCUMENT NUMBER: 119:63711

TITLE: Histamine activates chloride and potassium currents in guinea pig tracheal myocytes: convergence with muscarinic signalling pathway

AUTHOR(S): Janssen, Luke j.; Sims, Stephen M.

CORPORATE SOURCE: Dep. Physiol., Univ. Western Ontario, London, ON, N6A 5C1, Can.

SOURCE: Journal of Physiology (Cambridge, United Kingdom) (1993), 465, 661-77

CODEN: JPHYA7; ISSN: 0022-3751

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of histamine on membrane currents and contractile state of isolated guinea pig tracheal myocytes were examined using perforated patch and whole-cell recording techniques. The effects of histamine were

compared to those of acetylcholine (ACh) and caffeine. During voltage clamp ($V_{hold} = -60$ mV), histamine elicited contraction and an inward current (I_{hist}) which was often followed by current oscillations. I_{hist} had a reversal potential (V_{rev}) of -9 mV. I_{hist} was dependent on the Cl^- gradient and was antagonized by the Cl^- channel blocker niflumic acid. V_{rev} was more pos. (2 mV) when K^+ -selective currents were blocked by Cs^+ and $Et4N^+$. When all external Na^+ was replaced with N-methyl-D-glucamine, there was a small reduction in the amplitude of I_{hist} . The histamine-induced current was similar to that elicited by ACh and by caffeine with respect to time course, amplitude, and current-voltage relationship. Responses to histamine and to ACh were nonadditive, consistent with a convergence of histaminergic and cholinergic signalling pathways. I_{hist} was antagonized by the **H1 histaminergic receptor antagonist astemizole**, but not by atropine. When recorded using the perforated patch configuration, I_{hist} could be elicited repeatedly for >30 min. When cells were studied in the whole-cell configuration using a pipet solution containing 0.025 mM EGTA, the amplitude of I_{hist} was initially

the

same as that obtained using perforated patch but then decreased; the time required for the responses to decrease to 50% ($t_{1/2}$) was 8.2 min. When 1 mM EGTA was included in the pipet solution (whole-cell configuration), the initial response to histamine was decreased in size and $t_{1/2}$ was reduced to 3.3 min. The characteristics of the signalling pathway were examined in cells studied using a whole-cell configuration with 0.025 mM EGTA in the recording pipet. Heparin reduced $t_{1/2}$ to 4.3 min. GTPyS elicited inward current and oscillations; both effects were enhanced by histamine. GTPyS also reduced $t_{1/2}$ to 1.4 min. Pertussis toxin did not alter the amplitude or time course of I_{hist} . Thus, in guinea pig tracheal myocytes, binding of histamine to H1 receptors leads to **release** of Ca^{2+} from intracellular stores and subsequent activation of Cl^- and K^+ conductances as well as contraction. Furthermore, that ACh elicits similar physiol. responses due to a convergence of the histaminergic and muscarinic signalling pathways.

L32 ANSWER 10 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1993:551922 HCAPLUS

DOCUMENT NUMBER: 119:151922

TITLE: Acute and subchronic effects of the H1-
histamine receptor antagonist ebastine in 10 , 20 and 30 mg dose, and triprolidine 10 mg on car driving performance

AUTHOR(S): Brookhuis, K. A.; De Vries, G.; De Waard, D.

CORPORATE SOURCE: Traffic Res. Cent., Univ. Groningen, Haren, 9750 AB, Neth.

SOURCE: British Journal of Clinical Pharmacology (1993), 36(1), 67-70
CODEN: BCPHBM; ISSN: 0306-5251

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of a new antihistamine, **ebastine** (10 , 20 and 30 mg), on several parameters of driving performance in actual traffic were studied in 15 healthy male volunteers. Subjects were treated for 5 days, and their driving performance tested on day 1 and day 5. The study was double-blind, placebo controlled and included the antihistamine triprolidine (10 mg sustained **release**) as an active drug control. General tolerability was good except in one case following the reference compound triprolidine. No significant changes in driving performance were found with the new antihistamine **ebastine** at any dosage, on day 1 or day 5. Triprolidine (10 mg) significantly increased both the

amount of weaving and the delay in following speed maneuvers of a leading car, compared with placebo. The results suggest that **ebastine** in doses up to 30 mg may be relatively safe for use by those who drive motor vehicles while under medication. The results do not warrant such a conclusion for triprolidine at 10 mg.

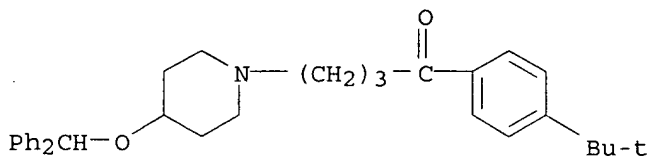
IT 90729-43-4, **Ebastine**

RL: BIOL (Biological study)

(driving skills response to, in humans)

RN 90729-43-4 HCAPLUS

CN 1-Butanone, 1-[4-(1,1-dimethylethyl)phenyl]-4-[4-(diphenylmethoxy)-1-piperidinyl]- (9CI) (CA INDEX NAME)



L32 ANSWER 11 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1986:122784 HCAPLUS

DOCUMENT NUMBER: 104:122784

TITLE: Modulation of in vitro anaphylaxis of guinea pig isolated tracheal segments by **azelastine**, inhibitors of arachidonic acid metabolism and selected antiallergic drugs

AUTHOR(S): Chand, N.; Diamantis, W.; Sofia, R. D.

CORPORATE SOURCE: Dep. Pharmacol., Wallace Lab., Cranbury, NJ, 08512, USA

SOURCE: British Journal of Pharmacology (1986), 87(2), 443-8

CODEN: BJPCBM; ISSN: 0007-1188

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The ability of **azelastine** [58581-89-8] to influence antigen-induced contractile responses (Schultz-Dale phenomenon) in isolated tracheal segments of the guinea-pig was investigated and compared with selected antiallergic drugs and inhibitors of arachidonic acid [506-32-1] metabolism. Indomethacin [53-86-1] produced a significant leftward shift of the antigen concentration-effect curve. The inhibitory activity of **azelastine** on anaphylactic responses in guinea-pig trachea was dependent on the duration of exposure (preincubation period). The relative order of potency (antianaphylactic activity) at calculated IC₅₀ level was as follows: FPL 55712 [40786-08-1] (a leukotriene receptor antagonist) > nordihydroguaiaretic acid [500-38-9] (a lipoxygenase inhibitor) > p-bromophenacyl bromide [99-73-0] (a phospholipase A₂ inhibitor) > BW 755c [66000-40-6] (a dual inhibitor of lipoxygenase and cyclooxygenase) > theophylline [58-55-9] (a phosphodiesterase inhibitor) > **azelastine** > diphenhydramine [58-73-1] (H₁ histamine-receptor antagonist) > ketotifen [34580-13-7] > cromoglycate [16110-51-3]. FPL 55712 (added 5 min before antigen challenge) was about 12 times as potent as **azelastine** (added 2 h before antigen challenge). The incubation of tracheal segments with **azelastine** and BW 755c for a period of 30 min inhibited indomethacin-augmented anaphylactic responses. These observations seem to suggest that **azelastine** and BW 755c interfere with the synthesis/release of the products of lipoxygenase/leukotriene

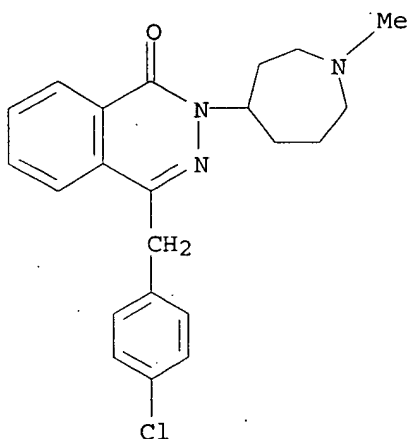
synthetase pathway (e.g., leukotrienes) in the mediation of allergic responses in airway smooth muscles.

IT 58581-89-8

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(antianaphylactic activity of, mechanism of, in trachea)

RN 58581-89-8 HCAPLUS

CN 1(2H)-Phthalazinone, 4-[(4-chlorophenyl)methyl]-2-(hexahydro-1-methyl-1H-azepin-4-yl)- (9CI) (CA INDEX NAME)



L32 ANSWER 12 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1985:482 HCAPLUS

DOCUMENT NUMBER: 102:482

TITLE: Histamine H1 receptor antagonists inhibit autoregulation of renal blood flow in the dog

AUTHOR(S): Banks, Robert O.; Inscho, Edward W.; Jacobson, Eugene D.

CORPORATE SOURCE: Coll. Med., Univ. Cincinnati, Cincinnati, OH, 45267, USA

SOURCE: Circulation Research (1984), 54(5), 527-35

CODEN: CIRUAL; ISSN: 0009-7330

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of H1 and H2 receptor antagonists on autoregulation of renal blood flow in the dog were studied. Renal arterial pressure was reduced in a step-wise fashion to 80 mm Hg by means of an adjustable aortic clamp positioned above the left renal artery. Infusion of H2 antagonists, cimetidine [51481-61-9] or ranitidine [66357-35-5] into the left renal artery at 10-5 mol/min had no effect on autoregulation of renal blood flow or on the reactive hyperemia that occurred when the aortic constriction was released. By contrast, intrarenal infusion of 10-5 mol/min chlorpheniramine maleate [113-92-8], an H1 receptor antagonist, reversibly attenuated reactive hyperemia and the ability of the kidney to autoregulate renal blood flow. Similar results were obtained with other, chemical dissimilar H1 antagonists (terfenadine [50679-08-8], diphenhydramine [58-73-1], and pyrilamine maleate [59-33-6]). The effects of chlorpheniramine on autoregulation of glomerular filtration rate also were evaluated. Before chlorpheniramine was infused (at 10-5 mol/min), the reduction of renal arterial pressure to 90 mm Hg had no effect on the glomerular filtration rate, whereas, during

infusion of the H1 antagonist, the glomerular filtration rate fell significantly when renal arterial pressure was reduced to 90 mm Hg. Infusion of histamine [51-45-6] (1 µg/kg per min) with increasing amts. of cimetidine, chlorpheniramine, diphenhydramine, or pyrilamine resulted in virtually identical dose-dependent decreases in histamine-induced renal vasodilation. However, even with 10⁻⁵ mol/min cimetidine or 10⁻⁵ mol/min chlorpheniramine, diphenhydramine, or pyrilamine, a significant histamine-induced renal vasodilation was observed. Thus, the amount of H1 antagonist required to inhibit histamine activation of H1 receptors is the same as needed to block autoregulation. Finally, renal vascular reactivity as estimated by acetylcholine-induced vasodilation was not substantially affected by chlorpheniramine or by pyrilamine. These observations provide evidence in support of a role for histamine as a chemical mediator of renal autoregulation.

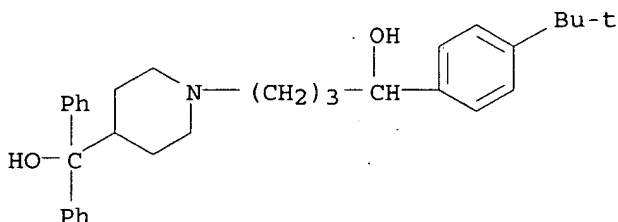
IT 50679-08-8

RL: BIOL (Biological study)

(kidney circulation autoregulation response to)

RN 50679-08-8 HCAPLUS

CN 1-Piperidinebutanol, α-[4-(1,1-dimethylethyl)phenyl]-4-(hydroxydiphenylmethyl)- (9CI) (CA INDEX NAME)



L32 ANSWER 13 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1982:504141 HCAPLUS

DOCUMENT NUMBER: 97:104141

TITLE: Performance studies with the H1-histamine
receptor antagonists,
astemizole and terfenadine

AUTHOR(S): Nicholson, A. N.; Stone, Barbara M.

CORPORATE SOURCE: R. Air Force Inst. Aviat. Med., Farnborough/Hants., UK

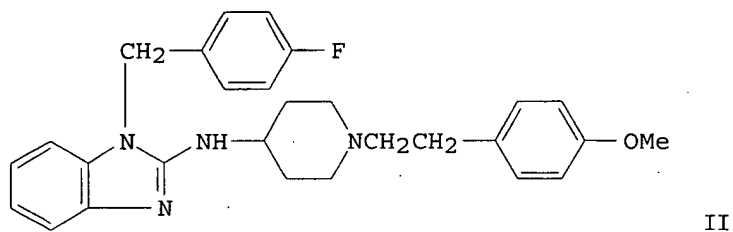
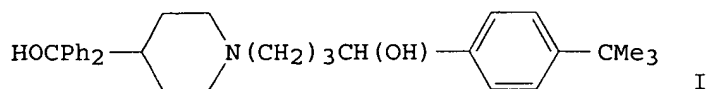
SOURCE: British Journal of Clinical Pharmacology (1982
, 13(2), 199-202

CODEN: BCPHBM; ISSN: 0306-5251

DOCUMENT TYPE: Journal

LANGUAGE: English

GI



AB Effects of the antihistamines, **terfenadine** (I) [50679-08-8] (60 mg) and **astemizole** (II) [68844-77-9] (10 and 20 mg), on performance (visuo-motor coordination, arithmetical ability and digit symbol substitution) and on mood were studied in 6 healthy adult females. The study was double-blind, placebo controlled, and included an antihistamine with known central effects (triprolidine [486-12-4] 10 mg in sustained **release** form). There were no changes in performance after **terfenadine** (60 mg) and **astemizole** (10 and 20 mg). Triprolidine (10 mg) caused a decrement in visuo-motor coordination 0.5 h after ingestion which lasted until 3.5 h. The subjects assessed their performance as impaired from 1.5-3.5 h with triprolidine (10 mg), and their mood assessments were also altered. **Terfenadine** and **astemizole** are likely to prove useful antihistamines for those involved in skilled activity.

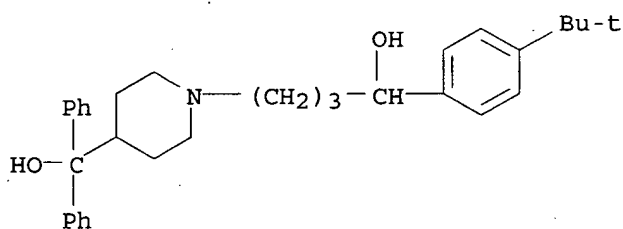
IT 50679-08-8 68844-77-9

RL: BIOL (Biological study)

(mental performance response to, in humans)

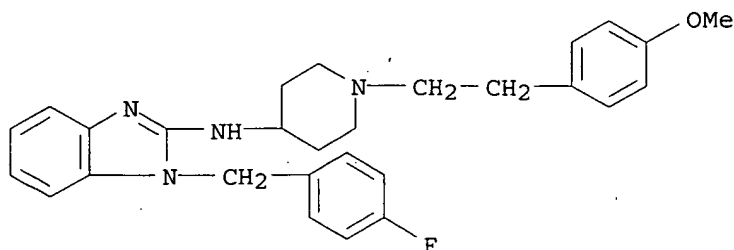
RN 50679-08-8 HCAPLUS

CN 1-Piperidinebutanol, α -[4-(1,1-dimethylethyl)phenyl]-4-(hydroxydiphenylmethyl)- (9CI) (CA INDEX NAME)



RN 68844-77-9 HCAPLUS

CN 1H-Benzimidazol-2-amine, 1-[(4-fluorophenyl)methyl]-N-[1-[2-(4-methoxyphenyl)ethyl]-4-piperidinyl]- (9CI) (CA INDEX NAME)



L32 ANSWER 14 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1979:149062 HCAPLUS

DOCUMENT NUMBER: 90:149062

TITLE: Gonadotropin-releasing activity in different parts of the rat brain and the probability of the existence of a gonadotropin-releasing hormone inhibiting factor in the suprachiasmatic area

AUTHOR(S): Savchenko, O. N.; Danilova, O. A.

CORPORATE SOURCE: Pavlov's Inst. Physiol., Leningrad, USSR

SOURCE: Fiziologicheskii Zhurnal SSSR imeni I. M. Sechenova (1979), 65(1), 111-16

CODEN: FZLZAM; ISSN: 0015-329X

DOCUMENT TYPE: Journal

LANGUAGE: Russian

AB Radioimmunochem. anal. of rat brain fragments indicated the presence of gonadotropin-releasing hormone (I) in relatively high amts. in the hypothalamic preoptic area, arcuate nucleus, median eminence, and retrochiasmatic area. I was detected in trace amts. in the hypothalamic suprachiasmatic area, mamillary bodies, and supraoptic, paraventricular, and ventromedial nuclei and in very low amts. in the pineal gland. No substantial sex differences were observed. The amts. of detectable I in combined retro- plus suprachiasmatic area fragments were lower than those in the retrochiasmatic area alone, suggesting that a I-inhibiting factor may be present in the suprachiasmatic area. Only the I levels in the preoptic area were significantly pos. correlated with the peripheral blood LH concns. (correlation coefficient = 0.583).

L32 ANSWER 15 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1975:544181 HCAPLUS

DOCUMENT NUMBER: 83:144181

TITLE: Secondary alkylsulfatases in a strain of Comamonas terrigena

AUTHOR(S): Fitzgerald, John W.; Dodgson, Kenneth S.; Matcham, George W. J.

CORPORATE SOURCE: Dep. Microbiol., Univ. Georgia, Athens, GA, USA

SOURCE: Biochemical Journal (1975), 149(2), 477-80

CODEN: BIJOAK; ISSN: 0264-6021

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Secondary alkylsulfatase activity towards K decan-5-yl sulfate at 30° was shown in C. terrigena. Cell-washing and osmotic shock procedures for releasing bacterial exocyttoplasmic enzymes were ineffective in releasing this activity. Primary alkylsulfatases could not be detected or induced in the organism.

L32 ANSWER 16 OF 16 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1972:415769 HCAPLUS

DOCUMENT NUMBER: 77:15769
TITLE: Comparative polyacrylamide electrophoresis of
periplasmic proteins **released** from
gram-negative bacteria by polymyxin B
AUTHOR(S): Cerny, G.; Teuber, M.
CORPORATE SOURCE: Inst. Bot., Tech. Univ., Munich, Fed. Rep. Ger.
SOURCE: Archiv fuer Mikrobiologie (1972), 82(4),
361-70

CODEN: ARMKA7; ISSN: 0003-9276

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Several cell-wall and membrane affecting agents were tested for causing **release** of periplasmic proteins of Escherichia coli B as compared by gel electrophoresis. Osmotic shock and polymyxin treatment yielded the best differentiated protein patterns. The periplasmic proteins derived from different E. coli strains and other gram-neg. bacteria by polymyxin treatment were compared. Whereas related strains showed similarities in the protein positions, unrelated gram-neg. bacteria showed great differences of the protein bands. The polymyxin-induced liberation of periplasmic proteins was dependent upon the growth phase and growth media of the bacteria and was severely inhibited by 10^{-2} M $MgCl_2$.

=> d que stat 128

L8 12 SEA FILE=REGISTRY ABB=ON (ACRIVASTINE OR ASTEMIZOLE OR AZELASTINE OR CETIRIZINE OR EBASTINE OR EPINASTINE OR FEXOFENADINE OR DESLORATADINE OR LORATADINE OR MIZOLASTINE OR NORASTEMIZOLE OR PROMETAZINE OR TERFENADINE)/CN

L9 1 SEA FILE=REGISTRY ABB=ON PROMETHAZINE/CN

L10 13 SEA FILE=REGISTRY ABB=ON L8 OR L9

L14 836 SEA FILE=HCAPLUS ABB=ON (?HISTAMINE?(W)?RECEPT?(W)?ANTAGON?)

L15 91 SEA FILE=HCAPLUS ABB=ON L14 AND (L10 OR ?ACRIVASTINE? OR ?ASTEMIZOLE? OR ?AZELASTINE? OR ?CETIRIZINE? OR ?EBASTINE? OR ?EPINASTINE? OR ?FEXOFENADINE? OR ?DESLORATADINE? OR ?LORATADINE? OR ?MIZOLASTINE? OR ?NORASTEMIZOLE? OR ?PROMETAZINE? OR ?TERFENADINE?)

L16 91 SEA FILE=HCAPLUS ABB=ON L14 AND L15

L18 13 SEA FILE=HCAPLUS ABB=ON L16 AND ?RELEAS?

L19 160 SEA FILE=HCAPLUS ABB=ON L16 OR ?OSMATIC?

L20 18 SEA FILE=HCAPLUS ABB=ON L19 AND ?RELEAS?

L21 18 SEA FILE=HCAPLUS ABB=ON L18 OR L20

L27 62 SEA L21

L28 34 DUP REMOV L27 (28 DUPLICATES REMOVED)

=> d ibib abs 128 1-34

L28 ANSWER 1 OF 34 MEDLINE on STN

ACCESSION NUMBER: 2006490128 IN-PROCESS

DOCUMENT NUMBER: PubMed ID: 16569656

TITLE: H1 histamine receptor antagonists induce genotoxic and caspase-2-dependent apoptosis in human melanoma cells.

AUTHOR: Jangi Shawkat-Muhialdin; Diaz-Perez Jose Luis; Ochoa-Lizarralde Borja; Martin-Ruiz Itziar; Asumendi Aintzane; Perez-Yarza Gorka; Gardeazabal Jesus; Diaz-Ramon Jose Luis; Boyano Maria Dolores

CORPORATE SOURCE: Department of Cell Biology and Histology, Faculty of Medicine and Dentistry, University of the Basque Country Leioa E-48940, Vizcaya, Spain.

SOURCE: Carcinogenesis, (2006 Sep) Vol. 27, No. 9, pp. 1787-96. Electronic Publication: 2006-03-28. Journal code: 8008055. ISSN: 0143-3334.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: NONMEDLINE; IN-DATA-REVIEW; IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 19 Aug 2006
Last Updated on STN: 19 Aug 2006

AB Previously, we found that the H1 **histamine receptor antagonist** diphenhydramine induces apoptosis in human acute T-lymphocytic leukemia cells. Since histamine has been shown to act as a growth factor in malignant melanoma cells, we decided to evaluate the in vitro effect of diphenhydramine and other H1 **histamine receptor antagonists**, such as **terfenadine**, **astemizol** and **triprolidine** on four malignant human melanoma cell lines. These antagonists were found to induce apoptotic cell death in all four melanoma cell lines. Apoptosis was determined by assessment of phosphatidylserine exposure on the surface of the cells and nuclear fragmentation. Importantly, H1 antagonist treatments did not adversely affect the viability of human melanocytes and murine fibroblasts at the same doses and duration of exposure. Treatment of melanoma cells with **terfenadine** induced DNA damage and caspases 2, 3, 6, 8 and 9

activation. Furthermore, the general caspase inhibitor (z-VAD-FMK) and a selective inhibitor of caspase-2 (z-VDVAD-FMK) protected melanoma cells from **terfenadine**-induced apoptosis. In contrast, the caspase-8 inhibitor (z-IETD-FMK) was ineffective. In addition, we found that mitochondria are involved in TEF-induced apoptosis, characterized by the dissipation of the mitochondrial transmembrane potential, the **release** of cytochrome c into the cytosolic compartment and caspase-9 activation. On the basis of these results we conclude that H1 **histamine receptor antagonists** induce apoptosis in human melanoma cells but not in normal melanocytes and embryonic murine fibroblasts; this apoptosis appears to be caspase-2-dependent and involves the mitochondrial pathway. The present results may contribute to the elaboration of novel therapeutic strategies for the treatment of malignant human melanoma.

L28 ANSWER 2 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005253679 EMBASE
TITLE: Roles of histamine and its receptors in allergic and inflammatory bowel diseases.
AUTHOR: Xie H.; He S.-H.
CORPORATE SOURCE: Prof. S.-H. He, Allergy and Inflammation Research Institute, Shantou University Medical College, 22 Xin-Ling Road, Shantou 515031 Guangdong Province, China. shoahenghe@hotmail.com
SOURCE: World Journal of Gastroenterology, (21 May 2005) Vol. 11, No. 19, pp. 2851-2857. .
Refs: 90
ISSN: 1007-9327 CODEN: WJGAF2
COUNTRY: China
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jun 2005
Last Updated on STN: 23 Jun 2005

AB Mast cell has a long history of being recognized as an important mediator-secreting cell in allergic diseases, and has been discovered to be involved in IBD in last two decades. Histamine is a major mediator in allergic diseases, and has multiple effects that are mediated by specific surface receptors on target cells. Four types of histamine receptors have now been recognized pharmacologically and the first three are located in the gut. The ability of **histamine receptor antagonists** to inhibit mast cell degranulation suggests that they might be developed as a group of mast cell stabilizers. Recently, a series of experiments with dispersed colon mast cells suggested that there should be at least two pathways in man for mast cells to amplify their own activation-degranulation signals in an autocrine or paracrine manner. In a word, histamine is an important mediator in allergic diseases and IBD, its antagonists may be developed as a group of mast cell stabilizers to treat these diseases. .COPYRG. 2005 The WJG Press and Elsevier Inc. All rights reserved.

L28 ANSWER 3 OF 34 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2005346108 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15997873
TITLE: Inhibition of tryptase **release** from human colon mast cells by **histamine receptor**

antagonists.

AUTHOR: He Shao-Heng; Xie Hua; Fu Yi-Ling
 CORPORATE SOURCE: Allergy & Inflammation Research Institute, Shantou University Medical College, Shantou 515031, China.. shoahenghe@hotmail.com
 SOURCE: Asian Pacific journal of allergy and immunology / launched by the Allergy and Immunology Society of Thailand, (2005 Mar) Vol. 23, No. 1, pp. 35-9. Journal code: 8402034. ISSN: 0125-877X.
 PUB. COUNTRY: Thailand
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200604
 ENTRY DATE: Entered STN: 7 Jul 2005
 Last Updated on STN: 14 Apr 2006
 Entered Medline: 13 Apr 2006

AB The main objective of this study was to investigate the ability of **histamine receptor antagonists** to modulate tryptase **release** from human colon mast cells induced by histamine. Enzymatically dispersed cells from human colon were challenged with histamine in the absence or presence of the **histamine receptor antagonists**, and the tryptase **release** was determined. It was found that histamine induced tryptase **release** from colon mast cells was inhibited by up to approximately 61.5% and 24% by the H1 **histamine receptor antagonist terfenadine** and the H2 **histamine receptor antagonist** cimetidine, respectively, when histamine and its antagonists were added to cells at the same time. The H3 **histamine receptor antagonist** clobenpropit had no effect on histamine induced tryptase **release** from colon mast cells at all concentrations tested. Preincubation of **terfenadine**, cimetidine or clobenpropit with cells for 20 minutes before challenging with histamine did not enhance the ability of these antihistamines to inhibit histamine induced tryptase **release**. Apart from **terfenadine** at 100 microg/ml, the antagonists themselves did not stimulate tryptase **release** from colon mast cells following both 15 minutes and 35 minutes incubation periods. It was concluded that H1 and H2 **histamine receptor antagonists** were able to inhibit histamine induced tryptase **release** from colon mast cells. This not only added some new data to our hypothesis of self-amplification mechanisms of mast cell degranulation, but also suggested that combining these two types of antihistamine drugs could be useful for the treatment of inflammatory bowel disease (IBD).

L28 ANSWER 4 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 2

ACCESSION NUMBER: 2003252815 EMBASE
 TITLE: Histamine alters E-cadherin cell adhesion to increase human airway epithelial permeability.
 AUTHOR: Zabner J.; Winter M.; Ashbourne Excoffon K.J.D.; Stoltz D.; Ries D.; Shasby S.; Shasby M.
 CORPORATE SOURCE: M. Shasby, Dept. of Internal Medicine, Univ. of Iowa College of Medicine, Iowa City, IA 52242, United States. michael-shasby@uiowa.edu
 SOURCE: Journal of Applied Physiology, (1 Jul 2003) Vol. 95, No. 1, pp. 394-401. .
 Refs: 23
 ISSN: 8750-7587 CODEN: JAPHEV

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
004 Microbiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Jul 2003
Last Updated on STN: 17 Jul 2003

AB During the immediate response to an inhaled allergen, there is an increase in the paracellular permeability of the airway epithelium. Histamine is an important agonist **released** during the immediate response to inhaled allergen. We hypothesized that histamine would increase human airway epithelial paracellular permeability and that it would do this by interrupting E-cadherin-based cell adhesion. Histamine, applied to the basolateral surface, increased the paracellular permeability of cultured human airway epithelia, and this effect of histamine was blocked by the **histamine receptor antagonist** promethazine. ECV304 cells express a histamine receptor, N-cadherin, and elements of the tight junction, including claudins, but they do not express E-cadherin. Histamine increased the paracellular permeability of ECV304 cells transfected with a vector and expressing E-cadherin but not ECV304 cells expressing lac-Z in the same vector. L cells do not express the histamine receptor, cadherins, or claudins. Histamine decreased adhesion of L cells expressing the human histamine receptor and E-cadherin to an E-cadherin-Fc fusion protein. Histamine did not alter the adhesion to the E-cadherin fusion protein of L cells expressing either the histamine receptor or E-cadherin alone. When applied to the apical surface, adenovirus poorly infects airway epithelial cells because its receptor, CAR, is restricted to the basolateral surface of the cells. When histamine was applied to the basolateral surface of airway epithelial cells, infection of the cells by adenovirus increased by approximately one log. This effect of histamine was also blocked by promethazine. Histamine increases airway paracellular permeability and increases susceptibility of airway epithelial cells to infection by adenovirus by interrupting E-cadherin adhesion.

L28 ANSWER 5 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003072580 EMBASE
TITLE: **Mizolastine** provides effective symptom relief in patients suffering from perennial allergic rhinitis: A double-blind, placebo-controlled study versus **loratadine**.
AUTHOR: Freche C.; Leynadier F.; Horak F.; Hide D.; Gracia F.D.; Goos M.; Bachert C.; Horvath A.; Antosova E.; Verrecchia M.; Soussen P.B.
CORPORATE SOURCE: Prof. F. Leynadier, Hopital Tenon, 4, rue de la Chine, 75970 Paris Cedex 20, France. francisque.leynadier@tnn.ap-hop-paris.fr
SOURCE: Annals of Allergy, Asthma and Immunology, (1 Sep 2002) Vol. 89, No. 3, pp. 304-310. .
Refs: 31
ISSN: 1081-1206 CODEN: ALAIF6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 27 Feb 2003
Last Updated on STN: 27 Feb 2003

AB Background: **Mizolastine** is a nonsedating H(1) **histamine receptor antagonist** with additional antiallergic properties currently marketed in Europe for the treatment of seasonal and perennial allergic rhinitis (PAR) and urticaria. Objective: This multicenter, randomized, double-blind, parallel-group study was conducted to evaluate the efficacy and safety of **mizolastine** in PAR compared with **loratadine** and placebo. Methods: After a 1-week placebo run-in period, 428 adult PAR patients received placebo (146 of 428), **mizolastine** 10 mg (141 of 428), or **loratadine** 10 mg (141 of 428) once daily for 28 days. Symptoms were evaluated by patients and physicians using a total nasal score, evaluating itching, rhinorrhea, nasal blockade, and sneezing severity. Results: **Mizolastine** treatment resulted in a significantly greater decrease in patient-rated total nasal score than placebo after 2 weeks (D14; -42%, $P < 0.001$) and at the end of the treatment period (-46%, $P = 0.01$), and significantly greater than that observed with **loratadine** at D14 ($P 0.031$). No significant difference in change in total nasal score was observed between **loratadine** and placebo at 2- and 4-week visits. The global safety was satisfactory and the incidence of adverse events was similar in the three treatment groups. Conclusions: **Mizolastine** provides effective symptom relief in PAR together with a satisfactory safety profile. Improvement with **mizolastine** was significantly greater than placebo throughout the study despite a large placebo effect. Also **mizolastine's** effects were greater those observed with **loratadine** after 2 weeks of treatment.

L28 ANSWER 6 OF 34 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2002016048 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11430478
TITLE: Roles of mast cells and histamine in mosquito bite-induced allergic itch-associated responses in mice.
AUTHOR: Ohtsuka E; Kawai S; Ichikawa T; Nojima H; Kitagawa K; Shirai Y; Kamimura K; Kuraishi Y
CORPORATE SOURCE: Department of Applied Pharmacology, Faculty of Pharmaceutical Sciences, Faculty of Medicine, Toyama Medical and Pharmaceutical University, Sugitani, Japan.
SOURCE: Japanese journal of pharmacology, (2001 May) Vol. 86, No. 1, pp. 97-105.
Journal code: 2983305R. ISSN: 0021-5198.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 21 Jan 2002
Last Updated on STN: 21 Jan 2002
Entered Medline: 7 Dec 2001

AB We investigated itch-associated responses (scratching) to mosquito bites and the role of histamine and mast cells in mosquito-induced itching in mice. Although the first bites of mosquito *Aedes albopictus* did not increase scratching, repeated bites increased scratching. The response was not diminished even after an interval of 2 months. Similarly, repeated intradermal (i.d.) injections of salivary gland extract (SGE) from *Aedes albopictus* increased scratching after SGE injection itself and mosquito bites. The scratching peaked within 10 min and almost subsided by 60 min. The opioid antagonist naloxone (1 mg/kg, s.c.) inhibited

scratching following SGE injection. Although the non-sedative H1-histamine-receptor antagonist **terfenadine** (30 mg/kg, p.o.) significantly suppressed scratching induced by histamine (100 nmol/site, i.d.) in either naive or mosquito-sensitized mice, it did not affect mosquito-induced scratching in mosquito-sensitized mice. Repeated injections of SGE increased scratching in mast cell-deficient (WBB6F1-W/Wv) mice as well as in normal (WBB6F1-+/+) littermates. Repeated exposure to mosquito bites roughly doubled serum concentrations of total IgE and IgG1, but not IgG2a. Repeated injections of SGE markedly increased plasma extravasation induced by mosquito bites and such an increase was almost completely suppressed by **terfenadine** (30 mg/kg, p.o.). The results show the presence of histamine-mediated and histamine-independent mechanisms in cutaneous itching and suggest that histamine probably **released** from mast cells does not play an important role in itching in immediate allergic reaction. Our murine model of mosquito itching may be useful for studying the mechanisms of immediate allergic itching.

L28 ANSWER 7 OF 34 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 ACCESSION NUMBER: 2000:473522 BIOSIS
 DOCUMENT NUMBER: PREV200000473522
 TITLE: The extracellular matrix molecule tenascin-R and its HNK-1 carbohydrate modulate perisomatic inhibition and long-term potentiation in the CA1 region of the hippocampus.
 AUTHOR(S): Saghatelian, Armen K.; Gorissen, Silke; Albert, Martine; Hertlein, Birgit; Schachner, Melitta [Reprint author]; Dityatev, Alexander
 CORPORATE SOURCE: Zentrum fuer Molekulare Neurobiologie, Universitaet Hamburg, Martinistrasse 52, D-20246, Hamburg, Germany
 SOURCE: European Journal of Neuroscience, (September, 2000) Vol. 12, No. 9, pp. 3331-3342. print.
 ISSN: 0953-816X.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 1 Nov 2000
 Last Updated on STN: 10 Jan 2002

AB Perisomatic inhibition of pyramidal cells regulates efferent signalling from the hippocampus. The striking presence of HNK-1, a carbohydrate expressed by neural adhesion molecules, on perisomatic interneurons and around somata of CA1 pyramidal neurons led us to apply monoclonal HNK-1 antibodies to acute murine hippocampal slices. Injection of these antibodies decreased GABAA receptor-mediated perisomatic inhibitory postsynaptic currents (pIPSCs) but did not affect dendritic IPSCs or excitatory postsynaptic currents. The decrease in the mean amplitude of evoked pIPSCs by HNK-1 antibodies was accompanied by an increase in the coefficient of variation of pIPSC amplitude, number of failures and changes in frequency but not amplitude of miniature IPSCs, suggesting that HNK-1 antibodies reduced efficacy of evoked GABA **release**. HNK-1 antibodies did not affect pIPSCs in knock-out mice deficient in the extracellular matrix molecule tenascin-R which carries the HNK-1 carbohydrate as analysed by immunoblotting in synaptosomal fractions prepared from the CA1 region of the hippocampus. For control, HNK-1 antibody was applied to acute sections of mice deficient in the neural cell adhesion molecule NCAM, another potential carrier of HNK-1, and resulted in decrease of pIPSCs as observed in wild-type mice. Reduction in perisomatic inhibition is expected to promote induction of long-term potentiation (LTP) by increasing the level of depolarization during theta-burst stimulation. Indeed, LTP was increased by HNK-1 antibody applied before stimulation. Moreover, LTP was reduced by an HNK-1 peptide mimic, but not control peptide. These results provide first evidence that

tenascin-R and its associated HNK-1 carbohydrate modulate perisomatic inhibition and synaptic plasticity in the hippocampus.

L28 ANSWER 8 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999250283 EMBASE
TITLE: Effect of antihistamines on epithelial cells.
AUTHOR: Devalia J.L.; Davies R.J.
CORPORATE SOURCE: Dr. J.L. Devalia, Academic Dept. of Respiratory Med., Royal London Sch. of Medicine/Dent., London Chest Hospital, Bonner Road, London E2 9JX, United Kingdom
SOURCE: Clinical and Experimental Allergy, Supplement, (1999) Vol. 29, No. 3, pp. 64-68. .
Refs: 20
ISSN: 0960-2178 CODEN: CLASEN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 2 Aug 1999
Last Updated on STN: 2 Aug 1999

AB Antihistamines have mostly been used in the management of allergic rhinitis, primarily for their symptomatic relief. Recent studies, however, have suggested that the non-sedating second-generation antihistamines also possess anti-inflammatory activity, and consequently may be useful in the management of inflammation in allergic airways disease. Several in vivo studies have demonstrated that antihistamines decrease inflammatory cell infiltration in allergic disease, mediator **release** from mast/basophil cells, and the expression of adhesion molecules on epithelial cells. Similarly, in vitro studies have demonstrated that antihistamines decrease the migration and activation of eosinophils and the **release** of pro-inflammatory mediators from mast/basophil cells, induced by immunological and non-immunological stimuli. More recent evidence suggests that the antihistamines may modulate airway inflammation by influencing the activity of airway epithelial cells, which due to their spatial arrangement and predominance in the airways, are thought to play a pivotal role in the aetiology of airway disease. We and others have demonstrated that antihistamines attenuate allergen- or chemical- induced expression and/or **release** of mediators which influence the activity of inflammatory cells, such as eosinophils, mast cells, basophils and lymphocytes, known to be involved in the pathogenesis of allergic airway diseases. Collectively, these studies suggest that second-generation H1- **histamine receptor antagonists** may have potential use either as safe anti- inflammatory alternatives to corticosteroids, or as rescue medication in combination with corticosteroids, for the management of severe airway disease.

L28 ANSWER 9 OF 34 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:312989 BIOSIS
DOCUMENT NUMBER: PREV199800312989
TITLE: Inhibition of leukotriene synthesis by **terfenadine** in vitro.
AUTHOR(S): Hamasaki, Y. [Reprint author]; Kita, M.; Hayasaki, R.; Zaitu, M.; Muro, E.; Yamamoto, S.; Kobayashi, I.; Matsuo, M.; Ichimaru, T.; Miyazaki, S.

CORPORATE SOURCE: Saga Med. Sch., Dep. Paediatr., 5-1-1 Nebeshima, Saga 849, Japan

SOURCE: Prostaglandins Leukotrienes and Essential Fatty Acids, (April, 1998) Vol. 58, No. 4, pp. 265-270. print.
CODEN: PLEAEU. ISSN: 0952-3278.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Jul 1998

Last Updated on STN: 22 Jul 1998

AB To determine the inhibitory mechanisms of **terfenadine** on the synthesis of leukotriene C4 (LTC4), an important mediator in allergic diseases, we evaluated the action of **terfenadine** on the IgE-dependent production of LTC4 in rat basophilic leukaemia 2H3 cells. Rat IgE-loaded cells were stimulated with anti-IgE in the presence or absence of various concentrations of **terfenadine** and the level of LTC4 **released** into the medium was measured by performing a specific radio immunoassay. **Terfenadine** inhibited the synthesis of LTC4 to 67.2% at a concentration of 5 mug/ml. LT synthesis was directly suppressed by inhibition of 5-lipoxygenase (5-LO) through calcium ion-independent mechanisms, and was also possibly suppressed by inhibition of cytosolic phospholipase A2 and 5-LO by blocking the influx of intracellular calcium ion that was initiated by IgE-related stimulation.

L28 ANSWER 10 OF 34 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:312360 BIOSIS

DOCUMENT NUMBER: PREV199799620163

TITLE: Highlights in cardiovascular effects of histamine and H-1-receptor antagonists.

AUTHOR(S): Genovese, A. [Reprint author]; Spadaro, G.

CORPORATE SOURCE: Div. Clinical Immunol. Allergy, Univ. Naples Federico II, Sch. Med., Via S. Pansini 5, 80131 Naples, Italy

SOURCE: Allergy (Copenhagen), (1997) Vol. 52, No. SUPPL. 34, pp. 67-78.

CODEN: LLRGDY. ISSN: 0105-4538.

DOCUMENT TYPE: Article

General Review; (Literature Review)

LANGUAGE: English

ENTRY DATE: Entered STN: 26 Jul 1997

Last Updated on STN: 26 Jul 1997

AB Despite numerous studies, the cardiac actions of histamine are still obscure. Yet, histamine could probably be clinically relevant. It is stored in large amounts in human cardiac tissue, where it is contained in the cytoplasmatic granules of mast cells (1). Mast cells are present in normal human heart tissue; they are more abundant in diseased human heart tissue where they lie in close proximity to blood vessels and between myocytes (2, 3). The histamine content of human heart mast cells is comparable to the histamine content of lung parenchymal and skin mast cells (4). Ultrastructural studies confirmed the presence of mast cells around vessels and between myocytes (2). Consequently, these cells are easily accessible to circulating antigens, drugs and stimuli that activate the cells to **release** vasoactive mediators which in turn can exert significant cardiovascular effects (5-7). Histamine possesses arrhythmogenic effects and once locally **released**, may enhance automaticity and induce triggering activity resulting in severe tachyarrhythmias. The major arrhythmogenic effects of histamine consist in increasing sinus rate and ventricular automaticity, and in slowing atrioventricular conduction (8). In addition, histamine may interfere with depolarization and repolarization through its effects on calcium and potassium currents. These effects are mediated by H-2-receptor (9).

Therefore direct activation of histamine receptor can induce cardiac arrhythmias. Consequently, the interference of these histaminergic effects may explain, at least in part, the arrhythmogenic effects described for some second-generation antihistamines, such as **terfenadine** and **astemizole** (10-25). In this brief review we will discuss the cardiac effects of histamine in experimental animal models and in man, and will review data on the safety of the new second-generation antihistamines, focusing on their cardiotoxic effects.

L28 ANSWER 11 OF 34 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 96350536 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8764989
TITLE: Evidence for bidirectional histamine transport by murine hematopoietic progenitor cells.
AUTHOR: Corbel S; Dy M
CORPORATE SOURCE: CNRS URA 1461, Hopital Necker, Paris, France.
SOURCE: FEBS letters, (1996 Aug 12) Vol. 391, No. 3, pp. 279-81.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 8 Oct 1996
Last Updated on STN: 8 Oct 1996
Entered Medline: 26 Sep 1996

AB Murine hematopoietic progenitor cells synthesize substantial amounts of histamine in response to IL-3 or calcium ionophore. They also take up extracellular histamine by an active transport system. In the present study we demonstrate that this system mediates both influx and efflux of histamine. Indeed, MR16155 and thioperamide, the two H3 antagonists which are most effective in inhibiting histamine uptake, likewise diminish the **release** of preloaded histamine from bone marrow cells. These compounds also inhibit the **release** of histamine which has been newly synthesized by hematopoietic progenitors in response to IL-3 or calcium ionophore, as assessed by the accumulation of the mediator inside the cells in the presence of the antagonists. The potency of different **histamine receptor antagonists** as inhibitors of histamine **release** increases with their capacity to block histamine uptake.

L28 ANSWER 12 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 5
ACCESSION NUMBER: 96268202 EMBASE
DOCUMENT NUMBER: 1996268202
TITLE: The contribution of histamine to the action of bradykinin in the human nasal airway.
AUTHOR: Austin C.E.; Dear J.W.; Neighbor H.; Lund V.; Foreman J.C.
CORPORATE SOURCE: Department of Pharmacology, University College London, Gower Street, London WC1E 6BT, United Kingdom
SOURCE: Immunopharmacology, (1996) Vol. 34, No. 2-3, pp. 181-189. .
ISSN: 0162-3109 CODEN: IMMUDP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 1 Oct 1996

Last Updated on STN: 1 Oct 1996

AB Bradykinin, 10 to 1000 µg given by aerosol into the nasal cavity of normal, healthy volunteers, produced a dose-related increase of nasal airway resistance. Bradykinin also reduced the minimal nasal cross-sectional area (A(min)), increased albumin **release** into nasal lavage fluid and increased the symptoms of nasal inflammation. Pretreatment with **cetirizine** (10 mg orally) reduced the fall in A(min) induced by bradykinin, 300 µg, but not by bradykinin, 100 µg. Pre-treatment of the subjects with the H1 **histamine receptor antagonist cetirizine** (10 mg, orally) or **terfenadine** (60 mg, orally) 3 h before bradykinin administration caused significant reduction of the bradykinin-induced increase in nasal airway resistance in the upper range of bradykinin doses (300-1000 µg) but not in the lower range (10-100 µg). **Cetirizine** reduced the albumin **release** into the nasal airway and the symptoms induced by bradykinin, 1000 µg. Following nasal challenge with bradykinin 300 µg or 1000 µg, no increase could be detected in the histamine content of nasal lavage fluid. Isolated human nasal cells **released** histamine in response to bradykinin, 33 and 100 µM, anti-IgE and calcium ionophore, A23187. We conclude that the actions of bradykinin in the human nasal airway are, in part, accounted for by the **release** of histamine.

L28 ANSWER 13 OF 34

MEDLINE on STN

DUPLICATE 6

ACCESSION NUMBER: 95304558 MEDLINE

DOCUMENT NUMBER: PubMed ID: 7785062

TITLE: Protective effect of various antagonists of inflammatory mediators against paraoxon-induced pulmonary edema in the rabbit.

AUTHOR: Delaunois A; Gustin P; Vargas M; Ansay M

CORPORATE SOURCE: Department of Pharmacology and Toxicology, Faculty of Veterinary Medicine, University of Liege, Belgium.

SOURCE: Toxicology and applied pharmacology, (1995 Jun) Vol. 132, No. 2, pp. 343-5.

Journal code: 0416575. ISSN: 0041-008X.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE).

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199507

ENTRY DATE: Entered STN: 26 Jul 1995

Last Updated on STN: 26 Jul 1995

Entered Medline: 17 Jul 1995

AB The protective effect of some antagonists of various inflammatory mediators against paraoxon-induced increases in endothelial permeability has been investigated in isolated perfused rabbit lungs. The edema induced by paraoxon has been previously related to a chain reaction mediated by acetylcholine. Lungs were ventilated and blood-free perfused with a constant flow. Arterial and venous pressures and lung weight were continuously recorded. Endothelial permeability was evaluated by measuring the capillary filtration coefficient (Kf,c). Paraoxon (4 x 10⁻⁴ M) was injected in the perfusion circuit, in lungs with or without pretreatment with atropine, ketanserin, clonidine, morphine, indomethacin, and **terfenadine** plus cimetidine. Paraoxon induced a time-dependent increase in the Kf,c, a maximal effect being recorded 60 min after the injection. All the antagonists used as pretreatment significantly reduced the maximal effect recorded after paraoxon. These results show that muscarinic receptor antagonists, inhibitors of

neuropeptides **release**, cyclooxygenase inhibitors, and 5-hydroxytryptamine and **histamine receptor antagonists** can protect the lung against the edema induced by paraoxon. This protective effect is due to inhibition of the chain reaction triggered by acetylcholine.

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ACCESSION NUMBER: 95050626 EMBASE
DOCUMENT NUMBER: 1995050626
TITLE: [Trends in the therapy of mastocytosis].
TRENDS IN DER THERAPIE VON MASTOZYTOSEN.
AUTHOR: Amon U.; Wehrhahn C.; Wolff H.H.
CORPORATE SOURCE: Klinik fur Dermatologie/Venerologie, Lubeck, Germany
SOURCE: H+G Zeitschrift fur Hautkrankheiten, (1995) Vol. 70, No. 1, pp. 61-67. .
ISSN: 0301-0481 CODEN: ZHKRAJ
COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: German
SUMMARY LANGUAGE: German; English
ENTRY DATE: Entered STN: 8 Mar 1995
Last Updated on STN: 8 Mar 1995

AB Mastocytosis refers to a spectrum of conditions of unknown etiology. It manifests itself in the form of tumour-like or diffuse hyperplasia of tissue mast cells. Mastocytosis may either involve the skin alone (e.g. mastocytoma, classical urticaria pigmentosa) or be presented as a systemic disease. The latter often includes mast cell infiltration of the gastrointestinal tract and the bone marrow. Aggressive or malignant forms are rare. Clinical manifestations may result from nonspecific activation of mast cells (due to physical trauma, drugs, toxins, food etc.) and subsequent local or systemic **release** of mast cell mediators. This leads to a variety of symptoms which in the severest cases can lead to shock reactions. Apart from excision of isolated mastocytomas, no curative therapy for mastocytosis is available. Besides avoidance of trigger factors, the treatment of choice for prophylaxis or therapy of unwanted reactions includes **histamine-receptor antagonists** and/or 'mast cell stabilizers'. The present review article summarizes modern therapeutic alternatives in patients with mastocytosis.

L28 ANSWER 15 OF 34 MEDLINE on STN DUPLICATE 7

ACCESSION NUMBER: 94118788 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7904710
TITLE: The differential effects of **histamine receptor antagonists** on morphine- and U-50,488H-induced antinociception in the mouse.
AUTHOR: Suzuki T; Takamori K; Takahashi Y; Narita M; Misawa M; Onodera K
CORPORATE SOURCE: Department of Pharmacology, School of Pharmacy, Hoshi University, Tokyo, Japan.
SOURCE: Life sciences, (1994) Vol. 54, No. 3, pp. 203-11.
Journal code: 0375521. ISSN: 0024-3205.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199402
 ENTRY DATE: Entered STN: 12 Mar 1994
 Last Updated on STN: 6 Feb 1998
 Entered Medline: 18 Feb 1994

AB The effects of thioperamide, an H3 antagonist, and histamine H1 and H2 antagonists (s.c.) on morphine (s.c. or i.c.v.)- and U-50,488H (i.c.v.)-induced antinociception in male ddY mice were examined using the hot-plate (55 degrees C) test. Thioperamide significantly inhibited morphine-induced antinociception, but not U-50,488H-induced antinociception. The suppressive effect of thioperamide on morphine-induced antinociception was reversed by the H1 antagonist pyrilamine, but not by the H2 antagonist zolantidine. On the other hand, pyrilamine significantly potentiated the antinociception induced by morphine, but not that induced by U-50,488H. Zolantidine significantly inhibited morphine-induced antinociception in a dose-dependent manner, but not U-50,488H-induced antinociception. Both **astemizole**, an H1 antagonist, and ranitidine, an H2 antagonist, which are known to barely cross the blood brain barrier, did not affect morphine-induced antinociception. These results suggest that morphine-induced antinociception may be potentiated by activation of H2 receptors and suppressed by activation of H1 receptors in the brain. Furthermore, neuronal histamine **release** induced by thioperamide may suppress morphine-induced antinociception through H1 receptors.

L28 ANSWER 16 OF 34 MEDLINE on STN DUPLICATE 8
 ACCESSION NUMBER: 94046665 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 8229856
 TITLE: Histamine activates Cl- and K+ currents in guinea-pig tracheal myocytes: convergence with muscarinic signalling pathway.
 AUTHOR: Janssen L J; Sims S M
 CORPORATE SOURCE: Department of Physiology, University of Western Ontario, London, Canada.
 SOURCE: The Journal of physiology, (1993 Jun) Vol. 465, pp. 661-77. Journal code: 0266262. ISSN: 0022-3751.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199311
 ENTRY DATE: Entered STN: 17 Jan 1994
 Last Updated on STN: 3 Mar 2000
 Entered Medline: 29 Nov 1993

AB 1. We investigated the effects of histamine on membrane currents and contractile state of isolated guinea-pig tracheal myocytes using perforated patch and whole-cell recording techniques. The effects of histamine were compared to those of acetylcholine (ACh) and caffeine. 2. During voltage clamp (Vhold = -60 mV), histamine elicited contraction and an inward current (Ihist) which was often followed by current oscillations. Ihist had a reversal potential (Vrev) of -9 +/- 3 mV. 3. Ihist was dependent on the Cl- gradient and was antagonized by the Cl- channel blocker niflumic acid. Vrev was more positive (+2 +/- 1 mV) when K(+)-selective currents were blocked by Cs+ and TEA. When all external Na+ was replaced with N-methyl-D-glucamine, there was a small reduction in the amplitude of Ihist. 4. The histamine-induced current was similar to that elicited by ACh and by caffeine with respect to time course, amplitude, and current-voltage relationship. Responses to histamine and to ACh were non-additive, consistent with a convergence of histaminergic and cholinergic signalling pathways. Ihist was antagonized by the H1

histaminergic receptor antagonist

astemizole, but not by atropine. 5. When recorded using the perforated patch configuration, Ihist could be elicited repeatedly for more than 30 min. When cells were studied in the whole-cell configuration using a pipette solution containing 0.025 mM EGTA, the amplitude of Ihist was initially the same as that obtained using perforated patch but then decreased; the time required for the responses to decrease to 50% ($t_{1/2}$) was 8.2 ± 1.0 min. When 1 mM EGTA was included in the pipette solution (whole-cell configuration), the initial response to histamine was significantly decreased in size and $t_{1/2}$ was reduced to 3.3 ± 0.7 min. 6. The characteristics of the signalling pathway were examined in cells studied using the whole-cell configuration with 0.025 mM EGTA in the recording pipette. Heparin significantly reduced $t_{1/2}$ to 4.3 ± 0.8 min. GTP gamma S elicited inward current and oscillations; both effects were enhanced by histamine. GTP gamma S also reduced $t_{1/2}$ to 1.4 ± 0.1 min. Pertussis toxin did not alter the amplitude or time course of Ihist. 7. We conclude that in guinea-pig tracheal myocytes, binding of histamine to H1 receptors leads to **release** of Ca^{2+} from intracellular stores and subsequent activation of Cl^- and K^+ conductances as well as contraction. Furthermore, we demonstrate that ACh elicits similar physiological responses due to a convergence of the histaminergic and muscarinic signalling pathways.

L28 ANSWER 17 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 9

ACCESSION NUMBER: 93165012 EMBASE

DOCUMENT NUMBER: 1993165012

TITLE: Influence of **histamine receptor**

antagonists on the dynamics of the cutaneous hypersensitivity reaction in patients infected with *Schistosoma haematobium*.

AUTHOR: Snyman J.R.; Sommers D.K.; Gregorowski M.D.

CORPORATE SOURCE: Department of Pharmacology, University of Pretoria, P.O. Box 2034, 0001 Pretoria, South Africa

SOURCE: European Journal of Clinical Pharmacology, (1993) Vol. 44, No. 5, pp. 467-471.

ISSN: 0031-6970 CODEN: EJCPAS

COUNTRY: Germany

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 004 Microbiology
013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 4 Jul 1993

Last Updated on STN: 4 Jul 1993

AB The biphasic cutaneous hypersensitivity response elicited by intradermal administration of *S. haematobium* antigen to patients with schistosomiasis may be used as a model for drug effects on cell dynamics. As the effects of H1- and H2-blockade, and the possible involvement of H3-receptors, have not been elucidated, we have examined the effects of combinations of **cetirizine**, cimetidine and betahistine on the response of patients with confirmed schistosomiasis. The skin blister technique was used. After intradermal administration of antigen, blister fluid containing inflammatory cells was collected on microscope slides at 6 and 24 h, and a differential cell count was done; and the area of induration was measured at 0.25, 1, 6 and 24 h. These baseline tests were repeated after 3 days of pretreatment with **cetirizine** 20 mg/d, after the addition of

cimetidine 1200 mg/d for 3 further days, and finally adding on betahistine 32 mg/d for 3 days. Simultaneous H1- and H2-blockade with citirizine plus cimetidine caused a significantly greater reduction in induration than citirizine (H1-blockade) alone; the reductions from the baseline value were 70%, 78%, 89%, 97%, and 33%, 53%, 43%, 30%, at times 0.25, 1, 6 and 24 h, respectively. The triple combination with the addition of betahistine (H1- and H2-agonist and H3-antagonist) resulted in reductions of 37%, 63%, 95% and 97% at the same times. The most striking changes in cellular dynamics were a significant increase in eosinophil (6 h) and neutrophil (6 h) vacuolation, and enhancement of monocyte (24 h) and basophil (6 h) accumulation, when the betahistine was added. Simultaneous H1- and H2-blockade significantly reduced eosinophil accumulation and vacuolation from baseline, and it also significantly inhibited progressive activation of neutrophils and eosinophils from 6 to 24 h when compared to baseline. As cell vacuolation and migration were increased when induration was minimal, we suggest that vascular effects were inhibited by H1- and H2-blockade, while other receptors, e.g. H3, regulated certain cellular effects. As histamine is known to cause immune modulation via H1- and H2-receptors, it is possible that the H3-receptor influences cell activity by a feedback mechanism, or perhaps by the **release** of other cytokines.

L28 ANSWER 18 OF 34 MEDLINE on STN DUPLICATE 10
 ACCESSION NUMBER: 93385010 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 8104017
 TITLE: Acute and subchronic effects of the H1-histamine
 receptor antagonist **ebastine** in
 10, 20 and 30 mg dose, and triprolidine 10 mg on car
 driving performance.
 AUTHOR: Brookhuis K A; De Vries G; De Waard D
 CORPORATE SOURCE: Traffic Research Centre, University of Groningen, Haren,
 The Netherlands.
 SOURCE: British journal of clinical pharmacology, (1993 Jul) Vol.
 36, No. 1, pp. 67-70.
 Journal code: 7503323. ISSN: 0306-5251.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: (CLINICAL TRIAL)
 Journal; Article; (JOURNAL ARTICLE)
 (RANDOMIZED CONTROLLED TRIAL)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199310
 ENTRY DATE: Entered STN: 5 Nov 1993
 Last Updated on STN: 6 Feb 1995
 Entered Medline: 21 Oct 1993
 AB 1. The effects of a new antihistamine, **ebastine** (10, 20 and 30
 mg), on several parameters of driving performance in actual traffic were
 studied in 15 healthy male volunteers. Subjects were treated for 5 days,
 and their driving performance tested on day 1 and day 5. The study was
 double-blind, placebo controlled and included the antihistamine
 triprolidine (10 mg sustained **release**) as an active drug
 control. 2. General tolerability was good except in one case following
 the reference compound triprolidine. No significant changes in driving
 performance were found with the new antihistamine **ebastine** at
 any dosage, on day 1 or day 5. Triprolidine (10 mg) significantly
 increased both the amount of weaving and the delay in following speed
 manoeuvres of a leading car, compared with placebo. 3. The results
 suggest that **ebastine** in doses up to 30 mg may be relatively
 safe for use by those who drive motor vehicles while under medication.
 The results do not warrant such a conclusion for triprolidine 10 mg.

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ACCESSION NUMBER: 1993:273758 BIOSIS
 DOCUMENT NUMBER: PREV199396003983
 TITLE: The effect of water stress on the net photosynthesis rate of six popular clones.
 AUTHOR(S): Liu, Jianwei; Liu, Yarong; Wang, Shiji
 CORPORATE SOURCE: Res. Inst. For., Chin. Acad. For., Beijing 100091, China
 SOURCE: Forest Research, (1993) Vol. 6, No. 1, pp. 65-69.
 ISSN: 1001-1498.
 DOCUMENT TYPE: Article
 LANGUAGE: Chinese
 ENTRY DATE: Entered STN: 9 Jun 1993
 Last Updated on STN: 9 Jun 1993

AB The effect of water stress by treating with PEG (MW 6000) solution with different osmotic potential (-0.4 MPa, -1.0 MPa, -1.6 MPa) on the net photosynthesis rate (P-n) of leaves of popular clones are studied in growth culture chamber. As for six clones of natured-drought, they are grouped into 3 fast- and 3 slower growing clones according to the significant difference in growth parameter. During 24 hours of various water stress, the P-n is obviously declined. After **release** from the water stress, the P-n of recovery of two growth types of clones shows that all clones survive in mild (-0.4 MPa), but not in moderate (-1.0 MPa) and severe (-1.6 MPa) **osmotic** stress at the end of two weeks. During stress and also after **release** of stress, the net photosynthesis rate of fast-growing clones decreased and also recovered faster than those of other slower growing clones, especially a faster-growing clone with water tolerant appearing the slowly dropping and fast recovering. P-n seems to be its significant growth contribution. This result will be helpful to identify the fast and drought tolerance clones in early-selection of poplar breeding.

L28 ANSWER 20 OF 34 MEDLINE on STN DUPLICATE 11

ACCESSION NUMBER: 89341180 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2503553
 TITLE: Inhaled lysine-aspirin as a bronchoprovocation procedure in aspirin-sensitive asthma: its repeatability, absence of a late-phase reaction, and the role of histamine.
 AUTHOR: Phillips G D; Foord R; Holgate S T
 CORPORATE SOURCE: Department of Immunopharmacology, Medicine I, Southampton General Hospital, England.
 SOURCE: The Journal of allergy and clinical immunology, (1989 Aug) Vol. 84, No. 2, pp. 232-41.
 Journal code: 1275002. ISSN: 0091-6749.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: (CLINICAL TRIAL)
 Journal; Article; (JOURNAL ARTICLE)
 (RANDOMIZED CONTROLLED TRIAL)
 LANGUAGE: English
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 198909
 ENTRY DATE: Entered STN: 9 Mar 1990
 Last Updated on STN: 3 Feb 1997
 Entered Medline: 21 Sep 1989

AB Inhalation of an aerosolized solution of lysine-aspirin has previously been described as a safer technique than oral challenge with aspirin for the diagnosis of aspirin-sensitive asthma. We describe a modification of this method that involves inhalation of serially doubling incremental concentrations of lysine-aspirin by a standardized technique and allows

construction of concentration-response curves. In 11 subjects with asthma, mean (SEM) age 48.2 (2.9) years, the geometric mean (range) provocation concentrations of histamine and lysine-aspirin required to produce a 20% decrease in FEV1 from baseline were 0.6 (0.04 to 3.2) and 48.3 (15.5 to 219) mg/ml, respectively. No relationship was found between these values. In seven of nine subjects investigated on two consecutive occasions, bronchoconstriction with lysine-aspirin was repeatable to within a single doubling concentration difference. Bronchoconstriction provoked by lysine-aspirin was more rapid than with oral aspirin and was not followed by any late asthmatic reaction or increase in nonspecific airway hyperresponsiveness. In six subjects, premedication with the selective H1 **histamine-receptor antagonist, terfenadine**, had no significant effect on bronchoconstriction provoked by inhaled lysine-aspirin, indicating little role for **release** of histamine in the response. We conclude that inhalation of lysine-aspirin may be used as a bronchoprovocation procedure for the diagnosis and investigation of aspirin-sensitive asthma.

L28 ANSWER 21 OF 34 MEDLINE on STN DUPLICATE 12
 ACCESSION NUMBER: 88146044 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 2894080
 TITLE: Effect of oral **terfenadine** on the
 bronchoconstrictor response to inhaled histamine and
 adenosine 5'-monophosphate in non-atopic asthma.
 AUTHOR: Phillips G D; Rafferty P; Beasley R; Holgate S T
 CORPORATE SOURCE: Medicine I, Southampton General Hospital.
 SOURCE: Thorax, (1987 Dec) Vol. 42, No. 12, pp. 939-45.
 Journal code: 0417353. ISSN: 0040-6376.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198804
 ENTRY DATE: Entered STN: 8 Mar 1990
 Last Updated on STN: 3 Feb 1997
 Entered Medline: 7 Apr 1988

AB Inhaled adenosine 5'-monophosphate (AMP) causes bronchoconstriction in atopic asthma, probably after in vivo conversion to adenosine. It has been suggested that adenosine potentiates preformed mediator **release** from mast cells on the mucosal surface of the airways by interacting with specific purinoceptors, without affecting the **release** of newly generated mediators. The airway response of nine non-atopic subjects with "intrinsic" asthma to inhaled AMP and the influence of the oral, selective H1 **histamine receptor antagonist terfenadine** on this response was investigated. The geometric mean provocation concentrations of histamine and AMP required to produce a 20% fall in FEV1 (PC20) were 1.82 and 13 mmol/l. In subsequent placebo controlled time course studies the FEV1 response to a single inhalation of the PC20 histamine was ablated after pretreatment with oral **terfenadine** 180 mg. This dose of **terfenadine** caused an 80% inhibition of the bronchoconstrictor response to the PC20 AMP when measured as the area under the time course-response curve and compared with the response to PC20 AMP preceded by placebo. **Terfenadine** 600 mg failed to increase protection against AMP further, but both doses of **terfenadine** delayed the time at which the mean maximum fall in FEV1 after AMP was achieved. **Terfenadine** 180 mg had no effect on methacholine induced bronchoconstriction in the same subjects. These data suggest that inhaled AMP may potentiate the **release** of preformed mediators from preactivated mast cells in the bronchial mucosa of patients with intrinsic

asthma.

L28 ANSWER 22 OF 34 MEDLINE on STN DUPLICATE 13
ACCESSION NUMBER: 87138885 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2880885
TITLE: Effect of **terfenadine** on methacholine-induced
bronchoconstriction in asthma.
AUTHOR: Patel K R
SOURCE: The Journal of allergy and clinical immunology, (1987 Feb)
Vol. 79, No. 2, pp. 355-8.
Journal code: 1275002. ISSN: 0091-6749.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198703
ENTRY DATE: Entered STN: 3 Mar 1990
Last Updated on STN: 29 Jan 1999
Entered Medline: 30 Mar 1987

AB The dose-response effect of nonsedating H1 **histamine-receptor antagonist, terfenadine**, administered orally in single doses, was studied on methacholine-induced bronchoconstriction in nine patients with extrinsic bronchial asthma in a double-blind, placebo-controlled, crossover trial. The doses of **terfenadine** used were 60 mg, 120 mg, and 180 mg, producing small but significant bronchodilator effect with all three doses at 2 hours. This response was still present at 4 hours. However, the provocative dose causing a 20% fall in FEV1 for methacholine was unaffected by all three doses of **terfenadine**. The bronchodilator response induced by antihistamines, including **terfenadine**, suggests an increased resting tone mediated by the constant presence of free histamine in the vicinity of H1 receptors in the airways and that methacholine acts directly on the airway muscarinic receptors and is not involved in local histamine release.

L28 ANSWER 23 OF 34 MEDLINE on STN DUPLICATE 14
ACCESSION NUMBER: 86160605 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3082402
TITLE: Modulation of in vitro anaphylaxis of guinea-pig isolated tracheal segments by **azelastine**, inhibitors of arachidonic acid metabolism and selected antiallergic drugs.
AUTHOR: Chand N; Diamantis W; Sofia R D
SOURCE: British journal of pharmacology, (1986 Feb) Vol. 87, No. 2, pp. 443-8.
Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198605
ENTRY DATE: Entered STN: 21 Mar 1990
Last Updated on STN: 21 Mar 1990
Entered Medline: 1 May 1986

AB The ability of **azelastine** to influence antigen-induced contractile responses (Schultz-Dale phenomenon) in isolated tracheal segments of the guinea-pig was investigated and compared with selected

antiallergic drugs and inhibitors of arachidonic acid metabolism. Indomethacin produced a significant leftward shift of the antigen concentration-effect curve. The inhibitory activity of **azelastine** on anaphylactic responses in guinea-pig trachea was dependent on the duration of exposure (preincubation period). The relative order of potency (antianaphylactic activity) at calculated IC₅₀ level was as follows: FPL 55712 (a leukotriene receptor antagonist) greater than nordihydroguaiaretic acid (a lipoxygenase inhibitor) greater than p-bromophenacyl bromide (a phospholipase A2 inhibitor) greater than BW 755c (a dual inhibitor of lipoxygenase and cyclo-oxygenase) greater than theophylline (a phosphodiesterase inhibitor) greater than **azelastine** greater than diphenhydramine (H1 **histamine-receptor antagonist**) greater than ketotifen greater than disodium cromoglycate. FPL 55712 (added 5 min before antigen challenge) was about 12 times as potent as **azelastine** (added 2 h before antigen challenge). The incubation of tracheal segments with **azelastine** and BW 755c for a period of 30 min was found to inhibit indomethacin-augmented anaphylactic responses. These observations seem to suggest that **azelastine** and BW 755c interfere with the synthesis/release of the products of lipoxygenase/leukotriene synthetase pathway (e.g., leukotrienes) in the mediation of allergic responses in airway smooth muscles.

L28 ANSWER 24 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 85112868 EMBASE
 DOCUMENT NUMBER: 1985112868
 TITLE: [Histamine-release induced by anaesthetic drugs or their solvents: Specific or nonspecific?].
 HISTAMINOLIBERATION INDUITE PAR LES PRODUITS ANESTHESIQUES OU LEURS SOLVANTS : SPECIFIQUE OU NON SPECIFIQUE?.

AUTHOR: Lorenz W.; Doenicke A.
 CORPORATE SOURCE: Department of Theoretical Surgery, Centre of Operative Medicine I, University of Marburg, 3550 Marburg/Lahn, Germany

SOURCE: Annales Francaises d'Anesthesie et de Reanimation, (1985) Vol. 4, No. 2, pp. 115-123. .
 CODEN: AFAREO

COUNTRY: France
 DOCUMENT TYPE: Journal
 FILE SEGMENT: 038 Adverse Reactions Titles
 024 Anesthesiology
 049 Forensic Science Abstracts
 037 Drug Literature Index
 026 Immunology, Serology and Transplantation

LANGUAGE: French
 SUMMARY LANGUAGE: English
 ENTRY DATE: Entered STN: 10 Dec 1991
 Last Updated on STN: 10 Dec 1991

AB Histamine release is a frequent event in the perioperative period. The reasons for its occurrence are complex; they include pseudoallergic and allergic phenomena - and probably a mixture of both. Some drugs and solvents seem to modulate histamine release induced by other drugs. Thus the terms 'specific and nonspecific' or 'selective and nonselective' histamine release which have their well appreciated place in experimental immunology and pharmacology should be avoided in describing histamine release responses in clinical conditions. The clinical relevance of histamine release in the perioperative period is considerable and can be compared with that of perioperative thrombosis and thromboembolism. Far too many drugs and

anaesthetic and surgical procedures give increased plasma histamine levels; premedication with H1- + H2-**histamine receptor antagonists** is therefore recommended in patients who have a history of hypersensitivity reactions to intravenous agents, a history of atopy, who are to be given the same drug a few days later, undergo surgery with a high risk of histamine **release**, are more than 70 years old or are poor risk patients with perioperative cardiac, respiratory or liver failure and shock.

L28 ANSWER 25 OF 34 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1986:26831 BIOSIS
DOCUMENT NUMBER: PREV198630026831; BR30:26831
TITLE: EFFECTS OF **HISTAMINE RECEPTOR ANTAGONISTS** OF NOREPINEPHRINE **RELEASE** AND REPERFUSION ARRHYTHMIAS IN THE ISOLATED RAT HEART.
AUTHOR(S): ROCHETTE L [Reprint author]; YAO-KOUME J; BRALET J; OPIE L H
CORPORATE SOURCE: FAC PHARMACY, DIJON, FR
SOURCE: Journal of Molecular and Cellular Cardiology, (1985) Vol. 17, No. SUPPL. 3.
Meeting Info.: 6TH MEETING OF THE INTERNATIONAL SOCIETY FOR HEART RESEARCH (EUROPEAN SECTION), STOCKHOLM, SWEDEN, SEPT. 8-11, 1985. J MOL CELL CARDIOL. CODEN: JMCDA. ISSN: 0022-2828.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 25 Apr 1986
Last Updated on STN: 25 Apr 1986

L28 ANSWER 26 OF 34 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 15

ACCESSION NUMBER: 85112741 EMBASE
DOCUMENT NUMBER: 1985112741
TITLE: Practical therapeutics: The use of **histamine receptor antagonists** in contemporary times.
AUTHOR: Obel A.O.K.
CORPORATE SOURCE: Division of Pharmacology and Therapeutics, College of Biological, Physical and Health Sciences, University of Nairobi, Nairobi, Kenya
SOURCE: East African Medical Journal, (1984) Vol. 61, No. 7, pp. 578-582.
CODEN: EAMJAV
COUNTRY: Kenya
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Pharmacology
026 Immunology, Serology and Transplantation
048 Gastroenterology
013 Dermatology and Venereology
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB Histamine is an autocoid of immense biological significance owing to its involvement in various physiological processes, allergic reactions, and response to injury. Its unbridled action may however aggravate injury and promote disease and human suffering. These latter features are

consequences of their exaggerated physiological actions secondary to enhanced release of histamine or augmented response to it or related autocoids. Antihistamines are beneficial in circumstances where histamine is primarily of pathological significance. Antihistamines may be of the H1 or H2 type. Both H1 and H2 antagonists are playing an increasingly crucial role in clinical medicine as well as elucidating the understanding of general biological processes. **Terfenadine** and **astemizole** are peripherally acting non-sedative antihistamines. Cimetidine is the 'gold standard' for H2 antagonists.

L28 ANSWER 27 OF 34 MEDLINE on STN
 ACCESSION NUMBER: 84261121 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 6204835
 TITLE: **Astemizole**. A review of its pharmacodynamic properties and therapeutic efficacy.
 AUTHOR: Richards D M; Brogden R N; Heel R C; Speight T M; Avery G S
 SOURCE: Drugs, (1984 Jul) Vol. 28, No. 1, pp. 38-61. Ref: 76
 Journal code: 7600076. ISSN: 0012-6667.
 PUB. COUNTRY: Australia
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198409
 ENTRY DATE: Entered STN: 20 Mar 1990
 Last Updated on STN: 6 Feb 1995
 Entered Medline: 12 Sep 1984

AB **Astemizole** is an H1-histamine receptor antagonist with a long duration of action permitting once daily administration. Its efficacy in seasonal and perennial allergic rhinitis has been convincingly demonstrated, and several comparative studies suggest that **astemizole** is at least as effective as some other H1-histamine receptor antagonists. A few smaller studies have shown beneficial effects on the symptoms of allergic conjunctivitis and chronic urticaria (but not atopic dermatitis). While **astemizole** appears to share with other H1-histamine receptor antagonists a tendency to increase appetite and cause weight gain after prolonged use, it offers the important advantage of an absence of significant central nervous system depression or anticholinergic effects with usual doses. Thus, **astemizole** offers a worthwhile improvement in side effect profile over 'traditional' H1-histamine receptor antagonists, especially in patients bothered by the sedative effects of these drugs.

L28 ANSWER 28 OF 34 MEDLINE on STN DUPLICATE 16
 ACCESSION NUMBER: 82135208 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 6120713
 TITLE: Performance studies with the H1-histamine receptor antagonists, **astemizole** and **terfenadine**.
 AUTHOR: Nicholson A N; Stone B M
 SOURCE: British journal of clinical pharmacology, (1982 Feb) Vol. 13, No. 2, pp. 199-202.
 Journal code: 7503323. ISSN: 0306-5251.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: (CLINICAL TRIAL)
 (CONTROLLED CLINICAL TRIAL)
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals

ENTRY MONTH: 198205
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 21 May 1982

AB 1 Effects of the antihistamines, **terfenadine** (60 mg) and **astemizole** (10 and 20 mg), on performance (visuo-motor coordination, arithmetical ability and digit symbol substitution) and on mood were studied in six healthy adult females. The study was double-blind, placebo controlled and included an antihistamine with known central effects (triprolidine 10 mg in sustained release form). 2 There were no changes in performance after **terfenadine** (60 mg) and **astemizole** (10 and 20 mg). Triprolidine (10 mg) caused a decrement in visuo-motor coordination (P less than 0.01) 0.5 h after ingestion which lasted until 3.5 h (P less than 0.001). The subject assessed their performance as impaired from 1.5-3.5 h (P less than 0.05) with triprolidine (10 mg), and their mood assessments were also altered. 3 **Terfenadine** (60 mg) and **astemizole** (10 and 20 mg) are likely to prove useful antihistamines for those involved in skilled activity.

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ACCESSION NUMBER: 81179091 EMBASE
DOCUMENT NUMBER: 1981179091
TITLE: Pre-scientific medicines: their extent and value.
AUTHOR: Scarpa A.
CORPORATE SOURCE: Ist. Italiano Etnomed., Rapallo, Italy
SOURCE: Social Science and Medicine - Part A Medical Sociology, (1981) Vol. 15, No. 3 II, pp. 317-326. .
CODEN: SMPSD8
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
036 Health Policy, Economics and Management
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB Pre-scientific medicines are immensely widespread involving about 80% of the world's population. This is a consequence of the connection between many pre-scientific medicines and religion, whereby many people resort to personal, domestic and popular medicines initially, because of the lack of any suitably scientifically trained staff. The environment, soil and climate, diversifies the various systems of pre-scientific medicine so that it is necessary to distinguish between those in arid zones, equatorial forests, cold climates and at great heights. The study of pre-scientific medicines has great implications for scientific research. Vast areas of research are available just in drugs, whether of animal, vegetable or mineral origin. Then there are the physical treatments, attention to the five sense-organs (melotherapy, coreotherapy, chromatotherapy, **osmatictherapy**, gustative and tactile sensations) which involve provocation of reflexes and releases of hormones, which can explain the success of many pre-scientific medicines. Then there are interesting therapies based on the psychic factors such as occurs in possession worship, trances, dreams, stress and emotional shocks. Certainly pre-scientific therapies have their own curative efficacy that stems from the phenomena described above. Pre-scientific medicines are no less important in the social field: the possibilities for information exchange between traditional and scientific medicine and the attempts to introduce pre-scientific medicines to other peoples who lack suitable scientific health assistance, because of the

absence of enough technically trained personnel, make pre-scientific medicines extremely valuable.

L28 ANSWER 30 OF 34 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1980:221564 BIOSIS
DOCUMENT NUMBER: PREV198070014060; BA70:14060
TITLE: ORIENTATION OF **ANOSMATIC** PIGEONS.
AUTHOR(S): PAPI F [Reprint author]; MARIOTTI G; FOA A; FIASCHI V
CORPORATE SOURCE: IST BIOL GEN, UNIV PISA, VIA A VOLTA 6, I-56100 PISA, ITALY
SOURCE: Journal of Comparative Physiology A Sensory Neural and Behavioral Physiology, (1980) Vol. 135, No. 3, pp. 227-232.
CODEN: JCPADN. ISSN: 0340-7594.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Test **releases** performed at 5 symmetrically arranged sites around the loft, at a distance of 78-99 km from it, showed that **anosmatics** birds transported without alteration of the earth's magnetic field were completely random-oriented; **anosmatics** birds transported in a container inside which the intensity of the magnetic field was strongly reduced were unable to orientate homewards and mostly departed according to a preferred compass direction; and control birds, which could smell and were transported without alteration of the magnetic field, were homeward oriented and performed better in homing than both experimental groups. **Anosmatics** birds may be unable to detect home direction at unfamiliar sites and magnetic stimuli perceived during the outward journey may be unable to substitute olfactory cues.

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ACCESSION NUMBER: 79008336 EMBASE
DOCUMENT NUMBER: 1979008336
TITLE: The effects of an antihistamine and/or a glucocorticoid on the prolactin response to surgical procedures.
AUTHOR: Chapler F.K.; Sherman B.M.; Swanson J.A.
CORPORATE SOURCE: Div. Reprod. Endocrinol., Dept. Obstet. Gynecol., Univ. Iowa Coll. Med., Iowa City, Ia., United States
SOURCE: American Journal of Obstetrics and Gynecology, (1978) Vol. 132, No. 4, pp. 367-372.
CODEN: AJOGAH
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
010 Obstetrics and Gynecology
003 Endocrinology
LANGUAGE: English

AB Prolactin (PRL) **release** in response to surgical stress has been demonstrated in a variety of species. Previous studies in rats indicate this response is blunted or blocked by pretreatment with either glucocorticoids or antihistamines. The present study was designed to investigate this phenomenon in man. Serum PRL levels before, during, and after major gynecologic surgery were measured in 20 women randomly assigned to one of four pretreatment regimens: dexamethasone, promethazine, both agents, and neither agent. Type of operation, preanesthetic medication, anesthetic agents, and estrogen status of patients were similar in all groups. Untreated controls exhibited the expected five- to tenfold increase in serum PRL concentration with surgery. Pretreatment with either dexamethasone or promethazine alone failed to suppress this response (in contrast to reported findings in the

rat) and in fact promethazine appeared to cause an augmented response. However, patients given dexamethasone and promethazine together exhibited only a two- to threefold PRL increase, a significantly lesser response than that in any of the other groups. Thus, PRL release in response to general anesthesia and surgery is inhibited by the combination of an antihistamine receptor antagonist and a glucocorticoid, whereas either agent alone has no suppressive effect.

L28 ANSWER 32 OF 34 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1979:197736 BIOSIS
DOCUMENT NUMBER: PREV197968000240; BA68:240
TITLE: DO AMERICAN AND ITALIAN PIGEONS RELY ON DIFFERENT HOMING MECHANISMS.
AUTHOR(S): PAPI F [Reprint author]; KEETON W T; BROWN A I; BENVENUTI S
CORPORATE SOURCE: IST BIOL GEN UNIV, VIA A VOLTA 6, I-56100 PISA, ITALY
SOURCE: Journal of Comparative Physiology A Sensory Neural and Behavioral Physiology, (1978) Vol. 128, No. 4, pp. 303-318. CODEN: JCPADN. ISSN: 0340-7594.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB In an attempt to explain the discrepancy between the results obtained by American and Italian investigators with reference to the role of olfaction in pigeon navigation, 5 series of homing experiments, most of which had already been performed on Italian pigeons, were conducted with Ithaca [New York, USA] birds. Application of an odorant (α -pinene) to the birds' beaks and nostrils did not produce consistent differences in initial orientation. Elimination of olfactory information during the outward journey did not result in different initial orientation. Pigeons made anosmatic by inserting plastic tubes in their nostrils were tested from familiar and unfamiliar release sites. At the familiar sites, the control bearings were randomly distributed and not homeward oriented. The experimentals, which were non-random in one site but random in the other, always were homeward oriented. At the unfamiliar site, all the groups were non-random in the other, always were homeward oriented. At the unfamiliar site, all the groups were non-randomly oriented. Homing performances were very different, the controls performing normally, the experimentals being mostly lost. Pigeons subjected to unilateral sectioning of the olfactory nerve and to plugging of 1 nostril; the ipsilateral in the control and the contralateral in the experimental birds, were tested from 2 unfamiliar sites. The orientation of the 2 treatments was nearly identical at both sites.

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ACCESSION NUMBER: 1976:197954 BIOSIS
DOCUMENT NUMBER: PREV197662027954; BA62:27954
TITLE: PURIFICATION AND SPECTROPHOTOMETRIC ASSAY OF NEOMYCIN PHOSPHO TRANSFERASE II.
AUTHOR(S): GOLDMAN P R; NORTHROP D B
SOURCE: Biochemical and Biophysical Research Communications, (1976) Vol. 69, No. 1, pp. 230-236. CODEN: BBRC9. ISSN: 0006-291X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable

AB Neomycin phosphotransferase II is maximally released by osmotic shocking of R+ Escherichia coli between late log and early stationary phase. A 300-400-fold purification of the enzyme protein is

accomplished by streptomycin sulfate and ammonium sulfate precipitations of osmotic shockates, followed by affinity and ion-exchange chromatography. The recovered enzyme preparation is electrophoretically 90% pure, is free of ATPase activity, and can be conveniently assayed spectrophotometrically by linking the production of ADP to pyruvate kinase and lactate dehydrogenase. The purified enzyme is not stable.

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ACCESSION NUMBER: 74199729 EMBASE

DOCUMENT NUMBER: 1974199729

TITLE: [Influence of H1 and H2 receptor antagonists on the effects of histamine in the circulatory system and on plasma histamine levels].

INTERNATIONAL SYMPOSIUM ON HISTAMINE

RECEPTOR ANTAGONISTS, LONDON, 19.

AUTHOR: Lorenz W.; Thermann M.; Hamelmann H.; et al.

CORPORATE SOURCE: Div. Exp. Surg. Pathol. Biochem., Surg. Clin., Univ. Marburg/Lahn, Germany

SOURCE: (1973) pp. 151-168. .

DOCUMENT TYPE: Book

FILE SEGMENT: 037 Drug Literature Index

030 Pharmacology

LANGUAGE: English

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

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(FILE 'HOME' ENTERED AT 14:32:14 ON 11 SEP 2006)

FILE 'HCAPLUS' ENTERED AT 14:32:54 ON 11 SEP 2006

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L1 314 SEA ABB=ON ("RICCI M"/AU OR "RICCI M A"/AU)
L2 1 SEA ABB=ON L1 AND ?NEURAMINIDASE?

FILE 'REGISTRY' ENTERED AT 14:34:30 ON 11 SEP 2006

FILE 'HCAPLUS' ENTERED AT 14:34:52 ON 11 SEP 2006

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L3 1 SEA ABB=ON 2004:267177/AN
SELECT RN L3 1-1

FILE 'REGISTRY' ENTERED AT 14:35:52 ON 11 SEP 2006

L4 66 SEA ABB=ON (9003-39-8/BI OR 100643-71-8/BI OR 102-76-1/BI OR
106392-12-5/BI OR 108612-45-9/BI OR 109-43-3/BI OR 110-17-8/BI
OR 124-07-2/BI OR 13463-67-7/BI OR 138-22-7/BI OR 139110-80-8/B
I OR 14807-96-6/BI OR 151-21-3/BI OR 153439-40-8/BI OR
162252-45-1/BI OR 1935-18-8/BI OR 204255-11-8/BI OR 24937-78-8/
BI OR 25086-89-9/BI OR 25322-68-3/BI OR 25322-69-4/BI OR
330600-85-6/BI OR 341969-96-8/BI OR 361-09-1/BI OR 39301-46-7/B
I OR 471-34-1/BI OR 475-31-0/BI OR 50-70-4/BI OR 50-81-7/BI OR
50-99-7/BI OR 503545-51-5/BI OR 50679-08-8/BI OR 56-81-5/BI OR
57-50-1/BI OR 57-55-6/BI OR 58581-89-8/BI OR 60-87-7/BI OR
623-50-7/BI OR 63-42-3/BI OR 68844-77-9/BI OR 69-65-8/BI OR
6915-15-7/BI OR 75970-99-9/BI OR 7647-14-5/BI OR 77-92-9/BI OR
7757-93-9/BI OR 79794-75-5/BI OR 80012-43-7/BI OR 81-25-4/BI
OR 83799-24-0/BI OR 83881-51-0/BI OR 87848-99-5/BI OR 9000-30-0
/BI OR 9001-67-6/BI OR 9002-96-4/BI OR 9004-32-4/BI OR
9004-34-6/BI OR 9004-35-7/BI OR 9004-38-0/BI OR 9004-65-3/BI
OR 9005-25-8/BI OR 9005-32-7/BI OR 9063-38-1/BI OR 90729-43-4/B
I OR 9085-05-6/BI OR 97-64-3/BI)

FILE 'HCAPLUS' ENTERED AT 14:36:12 ON 11 SEP 2006

L5 1 SEA ABB=ON L3 AND L4

FILE 'REGISTRY' ENTERED AT 14:40:32 ON 11 SEP 2006

L6 2 SEA ABB=ON (341969-96-8 OR 162252-45-1)/RN
L7 4 SEA ABB=ON (L6 OR ZANAMIVIR OR PERAMIVIR)
L8 12 SEA ABB=ON (ACRIVASTINE OR ASTEMIZOLE OR AZELASTINE OR
CETIRIZINE OR EBASTINE OR EPINASTINE OR FEXOFENADINE OR
DESLORATADINE OR LORATADINE OR MIZOLASTINE OR NORASTEMIZOLE OR
PROMETAZINE OR TERFENADINE)/CN
L9 1 SEA ABB=ON PROMETHAZINE/CN
D
L10 13 SEA ABB=ON L8 OR L9

FILE 'HCAPLUS' ENTERED AT 14:46:18 ON 11 SEP 2006

L11 16 SEA ABB=ON (?ANTIVIRAL? OR ?ANTI?(W)?VIRAL?) (W)?NEURAMINIDASE?
(W)?INHIBIT?
L12 124 SEA ABB=ON (?ANTIVIRAL? OR ?ANTI?(W)?VIRAL?) (W)?INHIBIT?
L13 1 SEA ABB=ON L12 AND (L7 OR ?ZANAMIVIR? OR ?PERAMIVIR?)
D AU
L14 836 SEA ABB=ON (?HISTAMINE?(W)?RECEPT?(W)?ANTAGON?)
L15 91 SEA ABB=ON L14 AND (L10 OR ?ACRIVASTINE? OR ?ASTEMIZOLE? OR
?AZELASTINE? OR ?CETIRIZINE? OR ?EBASTINE? OR ?EPINASTINE? OR

?FEXOFENADINE? OR ?DESLOTRADINE? OR ?LORATADINE? OR ?MIZOLASTI
NE? OR ?NORASTEMIZOLE? OR ?PROMETAZINE? OR ?TERFENADINE?)

L16 91 SEA ABB=ON L14 AND L15
L17 0 SEA ABB=ON L16 AND ?DUAL? (W) ?RELEAS?
L18 13 SEA ABB=ON L16 AND ?RELEAS?
L19 160 SEA ABB=ON L16 OR ?OSMATIC?
L20 18 SEA ABB=ON L19 AND ?RELEAS?
L21 18 SEA ABB=ON L18 OR L20
D AU 1-18
L22 0 SEA ABB=ON L19 AND (RICCI OR VERGEZ OR FAOUR)/AU
L23 0 SEA ABB=ON L19 AND VERGEZ
D L21 1-18
L24 0 SEA ABB=ON L16 AND (?OSMATIC? OR ?OSMOTIC?)
L25 0 SEA ABB=ON L14 AND (?OSMATIC? OR ?OSMOTIC?)
L26 14 SEA ABB=ON L21 AND (PRD<20030715 OR PD<20030715)

FILE 'MEDLINE, EMBASE, BIOSIS, JAPIO, JICST-EPLUS' ENTERED AT 14:58:38 ON
11 SEP 2006

L27 62 SEA ABB=ON L21
L28 34 DUP REMOV L27 (28 DUPLICATES REMOVED)
D AU 1-34

FILE 'USPATFULL' ENTERED AT 15:00:07 ON 11 SEP 2006

L29 115 SEA ABB=ON L21 AND (PRD<20030715 OR PD<20030715)
L30 55 SEA ABB=ON L29 AND (?OSMATIC? OR ?OSMOTIC?)
L31 2 SEA ABB=ON L30 AND ?DUAL? (3A) ?RELEAS?
D AU 1-2

FILE 'USPATFULL, HCAPLUS' ENTERED AT 15:02:33 ON 11 SEP 2006

L32 16 DUP REMOV L31 L26 HCAPLUS USPATFULL (0 DUPLICATES REMOVED)

FILE HOME

FILE HCAPLUS

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FILE COVERS 1907 - 11 Sep 2006 VOL 145 ISS 12

FILE LAST UPDATED: 10 Sep 2006 (20060910/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE REGISTRY

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 10 SEP 2006 HIGHEST RN 906318-57-8

DICTIONARY FILE UPDATES: 10 SEP 2006 HIGHEST RN 906318-57-8

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 30, 2006

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

FILE MEDLINE

FILE LAST UPDATED: 9 Sep 2006 (20060909/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details
on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_Mesh.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

FILE EMBASE

FILE COVERS 1974 TO 11 Sep 2006 (20060911/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default)
and biweekly.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

FILE BIOSIS

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 6 September 2006 (20060906/ED)

FILE JAPIO

FILE LAST UPDATED: 3 APR 2006 <20060403/UP>
FILE COVERS APRIL 1973 TO DECEMBER 22, 2005

>>> GRAPHIC IMAGES AVAILABLE <<<

>>> NEW IPC8 DATA AND FUNCTIONALITY NOT YET AVAILABLE IN THIS FILE.
USE IPC7 FORMAT FOR SEARCHING THE IPC. WATCH THIS SPACE FOR FURTHER
DEVELOPMENTS AND SEE OUR NEWS SECTION FOR FURTHER INFORMATION
ABOUT THE IPC REFORM <<<

FILE JICST-EPLUS
FILE COVERS 1985 TO 4 SEP 2006 (20060904/ED)

THE JICST-EPLUS FILE HAS BEEN RELOADED TO REFLECT THE 1999 CONTROLLED
TERM (/CT) THESAURUS RELOAD.

FILE USPATFULL
FILE COVERS 1971 TO PATENT PUBLICATION DATE: 7 Sep 2006 (20060907/PD)
FILE LAST UPDATED: 7 Sep 2006 (20060907/ED)
HIGHEST GRANTED PATENT NUMBER: US7103915
HIGHEST APPLICATION PUBLICATION NUMBER: US2006200885
CA INDEXING IS CURRENT THROUGH 5 Sep 2006 (20060905/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 7 Sep 2006 (20060907/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Jun 2006
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Jun 2006

=> log hold

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FULL ESTIMATED COST

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SINCE FILE

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